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Exposure and Risk Assessment for Arsenic (Revised)

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This report assesses the risk of exposure to arsenic. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in March of 1981.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of arsenic in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to arsenic for various subpopulations.

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March 1981
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AN EXPOSURE AND RISK ASSESSMENT
FOR ARSENIC

by

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FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

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1.0 TECHNICAL SUMMARY

1.1 INTRODUCTION

The Monitoring and Data Support Division, Office of Water Regulations and Standards, U.S. Environmental Protection Agency is conducting an ongoing program to identify the sources of and evaluate the exposure to 129 priority pollutants. This report assesses the environmental exposure to and risk associated with arsenic. Environmental releases of arsenic are both inadvertent, such as from fossil fuel combustion and metals production, and intentional from industries using arsenic in their products or processes. Arsenic is used in the manufacture of numerous pesticides, wood preservatives, glass, and various metals alloys, among other products.

1.2 RISK TO HUMANS: EXPOSURE, EFFECTS AND FATE CONSIDERATIONS

1.2.1 Cancer Risk Based on Taiwan Study

The risk associated with exposure of humans to adverse levels of arsenic in the environment is uncertain. Based on extrapolations from a Taiwan epidemiology study showing a relationship between skin cancer and low drinking water concentrations of total arsenic, risk estimates of excess individual lifetime skin cancer incidence associated with ingestion of 3 µg/l of arsenic in drinking water range from 0 to the range of 0.0009 to 0.0018. Three micrograms per liter is a commonly occurring arsenic concentration in water supplies. The implications of this very high rate of skin cancer for the U.S. must be qualified by several points: 1) attempts to induce tumors in laboratory animals exposed to arsenic have been unsuccessful; 2) the chemical form of arsenic in U.S. ground water may not be the same form as found in the Taiwanese study; 3) other potentially harmful chemicals were present in the Taiwan ground water (lysergic acid,ergotamines); 4) no significant increase in cancer was found in an epidemiology study of Lane County, Oregon in which relatively high arsenic levels are present in some of the water supply; 5) although the non-melanoma skin cancer rate is high in the U.S., the cancers tend to be located in different parts of the body than the Taiwanese cancers and there is a stong relationship between incidence rates and geographic latitude (i.e., ultraviolet radiation). Therefore, even without considering the inherently weak epidemiology studies themselves, there is a considerable amount of uncertainty associated with the extrapolation of the Taiwan skin cancer rate to the U.S. population.

Another important and largely unquantifiable source of uncertainty is in the application of the risk extrapolation models themselves. There is presently no scientific consensus as to which is the most appropriate model. Thus, the range of risk estimated by the various models may under-or overestimate the actual risk to man. Overestimation appears more likely due to the conservative assumptions utilized in the calculation of exposure and its duration.

1.2.2 Other Health Effects

The risk associated with exposure to environmental levels of arsenic is lower if one considers effects other than developing skin cancer that are reported for chronic exposure to low levels of various forms of arsenic in laboratory studies. Peripheral neuropathy resulted from exposure to the lowest reported effects level of 0.04 mg/kg/day of calcium arsenate ingested for 2-3 weeks. Other effects resulting from exposure to arsenate were observed at concentrations of 10 mg/kg or higher; concentrations of 23 mg/kg/day to 93 mg/kg/day did not elicit carcinogenesis in rats. Data for arsenite were very limited; skin disorders resulted from exposure to 0.13 mg/kg/day and no evidence of carcinogenesis was found in rats exposed to arsenite at levels of 13 mg/kg/day. Data for arsenic (unspecified), in addition to the Taiwan study, indicated central nervous system damage and lethality in infants exposed to 0.8 mg/kg/day and 7.9 mg/kg/day, respectively. The U.S. Interim Drinking Water Standard for total arsenic is 50 µg/l, which is equivalent to an intake level of 0.001 mg/kg/day from drinking water. In general the data base for understanding the toxicity of arsenic and its compounds is very poor.

1.2.3 Exposure Levels

Average exposure levels of arsenic for the general U.S. population through ingestion are less than 0.001 mg/kg for drinking water, total diet, and wine or moonshine. Consumption of contaminated fish or ground water may result in considerably higher levels exceeding 0.01 mg/kg/day. However, a low absorption efficiency of arsenic from ingestion of seafood has been reported. Dermal exposure for the majority of the U.S. population is expected to be negligible. Contact with arsenic-treated wood and contaminated detergent is associated with intake levels of 0.004 mg/kg/day and 0.06 mg/kg/day, respectively. However, the subpopulation exposed through these routes is small. Exposure of the general population through inhalation of ambient levels of arsenic is negligible and less than 0.001 mg/kg/day. Therefore the highest exposure levels for the general population are from ingestion routes.

Distinguishing between chemical forms of arsenic associated with different exposure routes is complicated due to a general lack of data, poor analytical techniques, and the presence of multiple forms in most media. However some exposure media with potentially high arsenite levels include well water, wine, and some fish tissue. Due to the uncertainty of these data risk is estimated for exposure to total arsenic levels. Therefore different exposure levels can be combined into exposure scenarios for selected subpopulations to better represent total exposure to arsenic.

The total estimated exposure to arsenic for the general non-smoking population and for children is approximately 0.4 µg/kg/day and 8 µg/kg/day, respectively. Smokers may be exposed to 1.7 µg/kg/day. Subpopulations with diets including ground water, fish, or wine highly contaminated with arsenic may be exposed to from 7 µg/kg/day to 143 µg/kg/day. Woodworkers may be exposed to from 4 µg/kg/day to 7 µg/kg/day. The worst case, assuming exposure to high but not the maximum levels of arsenic in all media, has an associated exposure of 41 µg/kg/day.

1.2.4 Risk Based on Other Studies

The risk of human exposure to adverse levels of arsenic is low for the general population, considering that the total intake level of 0.4 µg/kg/day is lower than the lowest available reported effect level for any arsenic compound of 40 µg/kg/day (peripheral neuropathy). In fact, most subpopulations have exposure levels lower than the acceptable intake level suggested by the U.S. Interim Drinking Water Standards. Subpopulations potentially at risk (with levels exceeding lowest effects levels) include people with large amounts of contaminated fish or ground water in their diets. The sizes of these subpopulations are unknown at this time due to limited information regarding the national distribution of ground water containing arsenic and of fish contamination.

A very limited amount of data is available on the toxicity of arsenic compounds to humans and laboratory animals. In addition, the available data do not always distinguish between chemical forms of arsenic nor does it adequately cover all the forms. The Taiwan study indicates adverse effects resulting from lower concentrations than toxic effects levels reported in other studies. Therefore, any conclusions made about the risk of humans in adverse exposure to arsenic are tentative and subject to change with additional data.

1.2.5 Relationship Between Environmental Fate of Arsenic and High Exposure Levels

There is a reasonable potential for ground water contamination by arsenic because of the element's ubiquity in solid waste discharge and the propensity of mobilization of some of its chemical forms under certain environmental conditions. Ground water is of particular concern for exposure because of the high efficiency of absorption of ingested amounts.

The potential sources of arsenic contamination of ground water drinking supplies, where it does exist, include: (1) fly ash disposal ponds distributed nationally, with 65% concentrated in the industrial Northeast; (2) pesticide use with 85% in the south-central and eastern U.S.; (3) production of other metals, especialy copper, iron and steel and boron, concentrated in the western states and industrialized areas in the East; (4) natural geological deposits of arsenic with unknown national distribution; local areas with such deposits have been identified in Oregon, California, Alaska, New Mexico and Nevada and are presumably scattered throughout the continent.

Surface drinking water supplies rarely contain excessive arsenic levels, even in towns with significant industrial sources of arsenic. This observation is compatible with the low magnitude of estimated releases made directly to surface water and the generally low ambient levels reported in STORET.

Food contamination can result from various environmental fate pathways for arsenic. Although most crops usually have low arsenic residues, root crops, leafy vegetables, and grapes used for wine sometimes have higher than average concentrations. Potential industrial sources of these residues include atmospheric fallout in the immediate vicinity of smelters, root uptake from soils contaminated by the disposal of solid waste from coal combustion, smelting and other industrial processes. However, this possibility is remote as crops are generally not cultivated in such areas. A potentially more significant source is arsenical pesticide use either near food crops or on soils previously contaminated where food crops are now grown. Natural background levels or arsenic may also be responsible for some contamination.

The likelihood of contamination of aquatic species which will be consumed by human populations is difficult to estimate because of a lack of information about national fish consumption patterns. Both marine and freshwater species, especially shellfish, living in waters with typical ambient concentrations can bioaccumulate tissue levels high enough to contribute to significant exposure levels in human consumers. However, there is evidence that only a small fraction of the amount ingested is actually absorbed. The total amount of arsenic intentionally discharged to surface waters is quite small. Inadvertent sources such as runoff from pesticide use, POTW discharges, urban runoff and natural contamination are probably responsible for the levels in surface waters in which aquatic life are exposed. Fate considerations applicable to the exposure of fish to adverse levels of arsenic are also relevant to bioaccumulation.

1.3 Risk to Nonhuman Biota: Exposure, Effects and Fate Considerations

Exposure to arsenic appears to present little risk to aquatic life. Freshwater acute toxicity can occur at sodium arsenite concentrations as low as 290 μg/l in the commonly found bluegill sunfish. Chronic toxicity data are limited to one study on daphnia finding approximately 900 μg/l to be toxic following long-term exposure. Ninety-nine percent of the concentrations were less than 100 μg/l in the 36,308 unremarked ambient samples reported in STORET over the past 5 years for total arsenic. A total of 51 observations were in excess of 1,000 μg/l and were reported in three major river basins: the Missouri, Upper Mississippi and the Colorado. A more detailed examination of the Missouri basin data indicated that the high levels were focused primarily in two areas, one associated with a mining facility and the other with no obvious sources nearby. There are no instances of fish kills on record within EPA directly attributed to arsenic.

Any apparent risk suggested by comparison of the monitoring data (for total As) and laboratory toxicity data (for soluble As) would be further modified by environmental parameters. Factors responsible for the retention and immobilization of arsenic in aquatic systems, such as the presence of hydrous oxides, clay sediment particulates, and so forth, would also tend to decrease the biological availability of the element.

The data on toxicity of arsenic to aquatic species are limited, so there is a possibility that species exist which are more sensitive to arsenic than those tested. Similarly, more currently available data are for the arsenite forms; there is a potential that other, more toxic, forms of arsenic may exist.

Exposure to soil levels of arsenic does not appear to present a significant risk to terrestrial plant and microbial species except in the highly contaminated soil immediately surrounding certain sources. Potential areas of contamination include: (1) soil downwind from smelters where atmospheric fallout is significant, (2) areas receiving arsenic pesticide applications, especially the inorganic forms, and, (3) areas high in natural background levels of arsenic.

1.4 Materials Balance of Arsenic

Nearly all known environmental releases of arsenic (43,000 kkg or 81%) are to land; releases to air and surface water account for 11% (5,700 kkg) and 8% (4,325 kkg), respectively, of the total releases made annually. The ratio of inadvertent to intentional releases is approximately 3.7 to 1 for all releases combined.

The atmosphere receives approximately 11% of all intentional releases of arsenic. Inadvertent sources such as fossil fuel combustion and metals production processes, were responsible for the largest fraction (70%) of the 5,700 kkg total arsenic loading to the atmosphere each year. Drift from pesticide use contributed 25% of all atmospheric releases.

Approximately 62% of the discharges to surface water were attributed to releases from POTWs and in urban runoff. The other important sources were runoff from pesticide applications to land and industrial discharge from zinc production. Effluent guidelines data indicated that industries with high wastewater concentrations of arsenic included nonferrous metals and ore mining operations, foundries, inorganic chemical producers, coal mining processes, printing/publishing and pulp/paper facilities.

Approximately 81% of the cultural releases of arsenic to the environment, from both inadvertent and intentional discharges, are made to land. Almost all sources of arsenic producers and consumers dispose of some form of arsenic-containing solid waste. The percentage of the total annual solid waste loading contributed by the most significant sources is as follows: 33% from fossil fuel combustion, 19% from pesticide use, 19% from copper production, 13% from iron and steel production and 5% from boron production. The remaining 11% is contributed by all other sources.

Less than 13% of the estimated 1,820 kkg of arsenic released each year in POTW effluents can be accounted for; known releases to treatment plants include veterinary chemical producers, a few metals production plants and urban runoff. Part of the remaining 87% may originate from natural loading and atmospheric particulate fallout. POTWs are not consistently effective at reducing arsenic concentrations in water, presumably due to the low effectiveness of primary and secondary treatment in removing arsenic.

2.0 INTRODUCTION

The Office of Water Regulations and Standards (OWRS), Monitoring and Data Support Division, of the U.S. Environmental Protection Agency is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated include potential harm to human beings and deleterious effects on fish and other biota. The goal of the task under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and to estimate the risk based on receptor exposure to these substances. The results are intended to serve as a basis for developing suitable regulatory strategy for reducing the risk, if such action is indicated.

This report is intended to provide a brief, but comprehensive, summary of the production, use, distribution, fate, effects, exposure, and potential risks of arsenic. Water-borne routes of exposure are stressed due to the emphasis of the OWRS on aquatic and water-related pathways. Occupational exposure and the exposure of the general population to atmospheric levels of arsenic are only considered in terms of the perspective they shed on the magnitude of water-related exposure.

There are a number of problems with attempting an exposure and risk assessment for this element. Since the purpose of this report is to provide a basis for regulation, it is important to identify those sources responsible for the majority of releases. Arsenic is an element commonly found in the earth's crust and natural sources to waterways can be significant. Thus in any analysis of discharges or runoff, it is important to distinguish background concentrations or natural sources from cultural sources. Releases from human activities include some known purposeful releases; however, the majority of arsenic discharges are unintentional releases resulting from smelting and coal combustion processes. Therefore, these estimates have a great deal of uncertainty associated with them.

Arsenic is found in the environment in numerous forms with differing fate characteristics, toxicology, and exposures. This exposure assessment focuses on the forms most prevalent in water: the inorganic forms of arsenate (pentavalent) and arsenite (trivalent), and various organic arsenic compounds (MSMA, DSMA, cacodylic acid) and, briefly, the arsines. Comparable information is not available for all forms and, in some cases, inferences must be made from information for similar forms. For example, data concerning concentrations of arsenic actually detected in environmental media are reported for "total arsenic" rather than for the individual chemical species. This lack of differentiation must be carried over in exposure estimates for receptors and precludes in some cases the drawing of conclusions regarding the toxic effects associated with exposure pathways and levels.

Although human epidemiologic data from Taiwan strongly implicate arsenic as a carcinogen to humans, no currently existing chronic laboratory animal studies support this observation. Due to the complexity of establishing dose-response relationships from epidemiologic studies, such as the inability to control all factors potentially contributing to carcinogenicity and differences between human subpopulations, the epidemiological data must be used cautiously. Risk extrapolations to the U.S. general population must be viewed in light of the limitations and assumptions of the analysis.

This report is organized as follows:

- Chapter 3.0 contains information on releases from the production, use, and disposal of arsenic, including identification of the form and amounts released and the point of entry into the environment.

- Chapter 4.0 considers the fate of arsenic in five specific pathways leading from point of entry into the environment until exposure of receptors and also reports available monitoring data of arsenic concentrations detected in environmental media.

- Chapter 5.0 discusses the adverse effects of arsenic and concentrations eliciting these effects in humans and quantifies the likely pathways and levels of human exposure to arsenic in various environmental media.

- Chapter 6.0 considers the effects of arsenic on non-humans and quantifies the environmental exposure of aquatic and terrestrial nonhuman biota to the element in different media.

- Chapter 7.0 discusses risk considerations for various subpopulations of humans and aquatic organisms, comparing estimated exposure levels of arsenic and the concentrations responsible for adverse effects.

- Appendices A, B, and C present the assumptions and calculations for the estimated environmental releases of arsenic described in Chapter 3.0. Appendix D includes information on the environmental distribution of arsenic releases.

3.0. MATERIALS BALANCE

3.1 INTRODUCTION

In this chapter, a materials balance is developed for arsenic. The materials balance identifies the initial point of entry of arsenic into the environment from all activities. Potential sources of releases were identified by a review of the numerous processes involving arsenic from its production or importation and use to its ultimate disposal.

For each major source of arsenic release, the amount of material released was estimated, and the environmental compartments (air, land and water) initially receiving and transporting the material were identified. Since arsenic has numerous chemical forms, the initial form of release was also identified. There are many uncertainties inherent in this type of analysis. Not all current releases have been identified, past releases were not well-documented, and future releases are difficult to predict. Nevertheless, sufficient information is available to indicate in general terms the nature, magnitude, and in some cases the location of pollutant loading of the environment with arsenic.

The scope of this chapter has been a review of both published and unpublished data concerning production, use, and disposal of arsenic within the United States. Available literature has been critiqued and compiled in order to present an overview of major sources of environmental release of arsenic and fully annotated tables to aid data evaluation.

3.2 MATERIALS BALANCE

In 1979, the total U.S. industrial demand for arsenic was approximately 14,640 kkg. Arsenic is produced within the U.S. and also imported. Inadvertent sources of arsenic from human activities are numerous, accounting for the annual release of 53,400 kkg of arsenic. Environmental releases during intentional and inadvertent production and from various consumptive uses of arsenic are presented in Table 3-1 and Figure 3-1. In addition, releases from POTWs and urban runoff are included.

The majority of environmental releases resulted from inadvertent production sources and from the use of arsenic as a pesticide. Purposeful production processes, POTWs, and urban runoff were responsible for lower, but significant, releases of arsenic. Other consumptive uses contributed a relatively small amount of arsenic environmental releases compared to other sources.

Table 3-1. Arsenic Releases from Production, Use, and Inadvertent Sources (kkg As, 1979)

Source	Quantity As	Estimated Environmental Releases				
				Water		
		Air	Land	Surface	POTW	Total[f]
PRODUCTION						
ASARCO, Tacoma[a]	6,700	210	1,200	<1	neg	1,410
USE[b]						
Pesticides	10,250	1,500	8,100	720		10,000
Wood Preservatives	2,930	neg	neg	neg		neg
Glass Manufacture	730	10	neg	neg		10
Alloys	440	c	c	c		
Other	290	2	10		<50	62
INADVERTENT SOURCES[d]						
Fossil Fuel Combustion[e]	17,000	2,000	14,000	150		17,000
Copper Production, 1° + 2°	9,900	1,100	8,100	38		9,300
Lead Production, 1° + 2°	990	230	1,100	neg		1,300
Zinc Production		280		560	1	830
Iron and Steel		55	5,700	9	6	5,700
Aluminum Production				180		180
Boron Production	2,250		2,200	4		2,200
Phosphorous Production	860		640	160		800
Manganese Production	4,000	10	1,400			1,440
Antimony Production			neg			neg
Cotton Ginning		300	580			880
POTW			20		1,800	1,820
Urban Runoff				1,050		1,050
TOTAL[f]		5,700	43,000	2,870	1,857	53,400

a) See Section 3.3.1 and Appendix A.

b) See Section 3 and Appendices A and B. Other includes electronics, catalysts, feed additives and veterinary chemicals. Use of arsenic-containing products is expected to release arsenic to the environment in the form as it is found in the product, i.e., use of MSMA would release arsenic to the environment as MSMA. Likewise, wood preserving would probably release arsenic as chromated copper arsenate or fluorchrome arsenate phenol. Glass and alloy manufacture would most likely release arsenic as the trioxide. The metallurgical inadvertent sources would probably also release arsenic as the trioxide, cotton ginning would release arsenic as the trioxide; cotton ginning would release arsenic as cacodylic acid (defoliant residue).

c) Included in advertent sources.

d) See Appendix A.

e) See Appendix C.

f) Values may not add due to rounding.

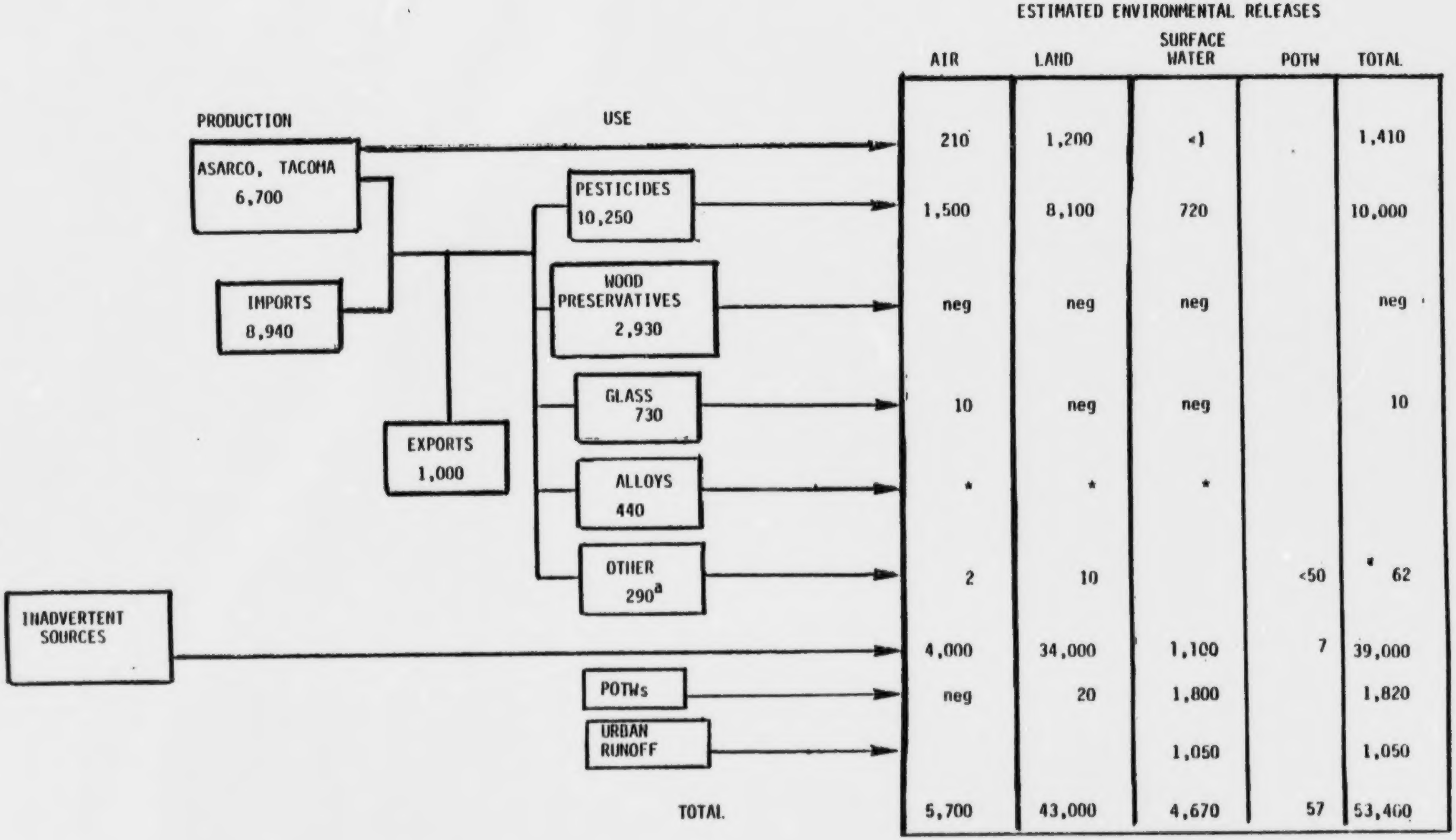
PRODUCTION
ASARCO, TACOMA 6,700
IMPORTS 8,940
EXPORTS 1,000
USE
PESTICIDES 10,250
WOOD PRESERVATIVES 2,930
GLASS 730
ALLOYS 440
OTHER 290a
INADVERTENT SOURCES
POTWs
URBAN RUNOFF
TOTAL
ESTIMATED ENVIRONMENTAL RELEASES
AIR
LAND
SURFACE WATER
POTW
TOTAL
210 1,200 <1 1,410
1,500 8,100 720 10,000
neg neg neg neg
10 neg neg 10
* * *
2 10 <50 62
4,000 34,000 1,100 7 39,000
neg 20 1,800 1,820
1,050 1,050
5,700 43,000 4,670 57 53,460

Figure 3-1. Environmental Releases of Arsenic: Production, Use, and Inadvertent Sources (kkg arsenic, 1979)

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a) Includes feed additives, veterinary chemicals, electronics, and catalysts.

*) Included in inadvertent sources, secondary metal production.

3.3 PRODUCTION AND INADVERTENT SOURCES

In 1979, about 6,420 kkg of arsenic were contained in the trioxide produced domestically; 280 kkg were recovered as arsenic metal (Nelson 1980). As noted in Table 3-2, approximately 8,940 kkg of arsenic were imported; 1,000 kkg were exported. Thus, the available domestic supply totaled 14,640 kkg arsenic.

3.3.1 Production

Arsenic trioxide (As_2O_3) and arsenic "metal" are recovered only at one facility in the U.S. The ASARCO smelter in Tacoma, Washington, produces arsenic from flue dust captured during the smelting of copper ore. Although arsenic is a minor constituent of most nonferrous ores, their low arsenic content does not warrant recovery.

Arsenic trioxide is volatilized during smelting and concentrates in flue dusts. The dusts are mixed with pyrite or galena and roasted; vapors from the roasting process are cooled and condensed in a series of brick chambers yielding a 90-95% pure arsenic trioxide (EPA 1979g). The product is removed by chipping the solid condensate from the brick walls. Resublimation in a reverberatory furnace and recondensation provide a product of even higher purity (EPA 1976a). To produce arsenic metal, the trioxide is reduced with carbon (EPA 1979g).

3.3.1.1 Environmental Releases of Arsenic from Production

Of the total 1,410 kkg arsenic released to the environment from arsenic production, 15% (210 kkg) is emitted to the atmosphere and 85% (1,200 kkg) is disposed of on land (see Table 3-2). Atmospheric emissions are largely due to particulates that escape control devices and, to a lesser extent, transfer and handling of materials (EPA 1979a,b). Land-destined arsenic is chiefly contained in slag (see Appendix A, Notes 1 and 2).

Arsenic recovery is a dry operation; wastewater occurs only from daily wash down of the facility. Based on a daily flow of 3,400 liters containing 310 mg arsenic per liter and assuming a 365 day per year operation, only 0.4 kkg of arsenic would be discharged from the production site per year (EPA 1979b, EPA 1975a). Such waters are directly discharged (EPA 1975a).

3.2.2 Inadvertent Sources

Arsenic is a constituent of most mineral ore; processing of these ores, as well as smelting and refining of the metal, releases substantial quantities of arsenic to the environment. Cotton ginning and disposal of the resulting wastes also introduce arsenic into the environment in the form of arsenical defoliant residue. Combustion of fossil fuel for space heating, electricity generation, and transportation releases arsenic to the environment as arsenic trioxide condensate on particulates and as arsenic-containing ash disposed in sluice ponds. Derivations of quantities of arsenic released to the environment via fossil fuel combustion

Table 3-2. Production of Arsenic, 1979 (kkg)

Production		Quantity[a]	Estimated Environmental Releases				
					Water		
			Air	Land	Surface	POTW	Total
ASARCO, Tacoma[b]	As_2O_3	6,420	210[g]	1,200[h]	<1[f]		1,410
	As[d]	280					
Imports[c]		8,940	----------neg---------				
Exports[e]		1,000	----------neg---------				
Available U.S. Supply		14,640					

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a) Quantity is kkg of As, figures are rounded nearest 10 kkg.

b) Nelson, 1980

c) Loebenstein, 1980. Based on weight % As of the imported arsenic containing compounds: Arsenic trioxide 11,180 kkg (75.7%) arsenic metal 370 kkg (100%), arsenic sulfide 35 kkg (61%), sodium arsenate 1 kkg (39%), arsenic acid 160 kkg (53%) arsenic compounds, not elsewhere classified (wood preservatives) 1 kkg (50%). Loebenstein, 1980b. Releases from processing of imported material are included in the total production/release estimates for the ASARCO, Tacoma facility.

d) Nelson 1980.

e) Loebenstein, 1980c.

f) EPA, 1979b, EPA 1975a. As_2O_3/As production is a dry operation. Wastewater is discharged at a rate of 2,270 to 3,400 l/day from washing down the plant; the water contains 310 mg As per liter (EPA, 1975). Therefore, as a maximum raw effluent estimate 3,400 l/day x 310 mg As/l x 365 day/yr operation: 0.38 kkg As would be discharged per year. EPA, 1979b states that their are no indirect dischargers in the primary copper industry so the estimated 0.38 kkg would be sent to surface waters.

g) Based on EPA, 1979a; 1979b. Approximately 45 kkg of the total 460 kkg of As emissions was a result of fugitive emissions; about 166 kkg escape control devices on the stack. See Appendix A, Note 1.

h) Based on EPA 1979a; 1976b, and EPA, 1975. See Appendix A, Note 2.

are found in Appendix C.* As noted in Tables 3-3 and 3-4, approximately 87% (34,147 kkg) of the total arsenic released from inadvertent sources is contained in land-disposed metal processing slag, 10% (4,000 kkg) is emitted to the atmosphere, and 2% (1,100) is discharged to water (derivations of these quantities are found in Appendix A).

Arsenic is contained in most metal products, and as such, is not released to the environment from ordinary usage. However, arsine gas can be formed when arsenic-containing metal comes in contact with acid (i.e., pickling of metal) (EPA 1976a). Also, arsenic associated with phosphorous, ultimately utilized in detergents, is apparently released to waterways. Angino et al. (1979) found that the 1-70 mg/kg arsenic in various detergents and presoaks yielded 1-250 mg/l arsenic in washwater.

3.4 USES OF ARSENIC

Current industrial uses of arsenic include pesticides, wood preservatives, additives in glass manufacture, nonferrous alloys, and feed additives; arsenic finds minor use in the electronics and chemical/catalysts industries. Discontinued uses include leather tanning chemicals and paint pigment additives (EPA 1976a). Demand for arsenic is expected to increase at an annual rate of 1% through 1985 (Loebenstein 1980).

As is the case with arsenic production data, consumption data are considered proprietary information. The use distribution pattern presented in Table 3-5 is based on previous years' consumption data and Bureau of Mines estimates (Loebenstein 1980). By far, the largest use of arsenic (70% or 10,250 kkg) is in pesticide manufacture.

3.4.1 Production and Use of Pesticides

Arsenic is a component of insecticides (Paris green, lead arsenate, calcium arsenate), herbicides (disodium methanearsenate, monosodium methanearsenate, cacodylic acid) and defoliants (arsenic acid). Table 3-6 gives quantities of arsenic contained in these pesticides in 1979, as well as environmental releases from the pesticides' production and use. Process flow diagrams are presented in Appendix B; producers and their locations are given in Table B-1.

3.4.1.1 Monosodium Methanearsenate (MSMA) and Disodium Methanearsenate (DSMA)

DSMA and MSMA are used as contact herbicides on weeds which are especially difficult to control. Both are registered for directed application (no contact with crop plants) on citrus fruits and cotton. DSMA is produced by reacting arsenic trioxide with sodium hydroxide, then adding methyl chloride (EPA 1975d). MSMA is then manufactured by treating DSMA with sulfuric acid.

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*Estimates of the national annual arsenic emissions from energy production range from 650 kkg/yr to 4,600 kkg/yr with the variation due to coal usage figures and the concentration of arsenic in coal which is highly variable across the country (see Note p.4-58). It should be noted that quantification of exposure based on these levels would give significantly different results depending upon the estimates used.

Table 3-3. Inadvertent Sources of Arsenic, 1979 (kkg)

Source	Input	Contained	Estimated Environmental Releases				
					Water		
			Air	Land	Surface	POTW	Total
Fossil Fuel Combustion (total)			2,000	14,000	150		17,000
Coal (total)[a]	16,450		2,000	14,000	150		17,000
Petroleum[b]	76		74	2	neg		76
Copper Production 1°[c]	9,850	3,070	1,100	6,730	44		7,874
2°[d]		neg	neg		neg	neg	
Lead Production 1°[e]	990	20	230	760	neg	neg	990
2°[f]			neg	300	neg	neg	300
Iron and Steel Production[g]		79,000	55	5,700	9	6	5,700
Zinc Production[h]			280		560	1	830
Aluminum Production[i]					180		180
Boron Production[j]	2,250	20		2,200	4		2,200
Phosphorus Production[k]	860			640	160		800
Manganese Production[l]	4,000	2,160	10	1,400			1,440
Antimony Production[m]				neg			neg
Cotton Ginning[n]			300	580			880
TOTAL			4,000	32,000	1,100	7	38,100

Footnotes next page.

Table 3-3. (Continued)

a) Includes external combustion and space heating. See Appendix C for release by type of coal and combustion. Water value is acid mine drainage and ashponds (94 kkg from mine drainage, 52 from ash ponds); Land value includes particulate >3 µm which settles to land. See Table 3-4. EPA, 1976a, 1975a. Blanks indicate data not available.

b) See Appendix C.

c) EPA, 1979a, 1975c. See Table A-3 and Appendix A, Note 6. Includes 1,400 kkg As sent to land as refinery slag, the chief source of As release. Also includes arsenic release from copper mining and milling (see Table 3-4) from copper mining, (see Table 3-4) 110 kkg As to air; 630 kkg As to land; 14 kkg As to surface water. EPA, 1976a. See Table 3-4.

d) Based on 50 secondary copper smelters, average As effluent of 0.02 kkg As/yr/plant. EPA 1975b. See Appendix A, Note 7. Arsenic concentration in secondary copper smelter feed material is 30-1000 times less than that for primary smelters, emission factors are about 100 times less than primary; atmospheric emissions are negligible. Arsenic in slag from secondary copper production total about 300 kkg. See Appendix A, Note 19. EPA, 1980b.

e) Atmospheric emissions based on 581,600 kkg lead produced (Rathjen, 1980), as an emission factor of 0.4 kg As/kkg Pb (Davis, 1971). Land discharges based on 410 kg slag and 40 kg dry sludge/kkg lead produced and an arsenic content of 0.29% EPA, 1976a. Wastewater discharge based on average As concentration for 4 plants, and a total of 7 plants, see Appendix A, Note 8. Contained quantity based on 581,600 kkg produced x 35g As/kkg lead produced, EPA, 1976a.

f) EPA, 1980b. Atmospheric emissions are negligible due to low arsenic content (0.4%) and processing temperatures (260-370°C) which are lower than the vaporization temperature of arsenic (613°C). Alternatively, applying the 0.4 kg As/kkg lead produced emission factor and 778,930 kkg lead produced from second smelters, 310 kkg As are released per year. Wastewater data from EPA, 1979b. Based on 69 plants x 8.0×10^{-3} kkg As/plant/yr. See Appendix A, Note 9. Land discharges based on 148,300 kkg slag/yr from secondary blast furnaces and 0.2% As in slag, EPA, 1976a.

g) See Appendix A, Note 18. Includes 3 kkg As omitted to air from ore mining, see Table 3-4.

h) Wastewater values based on an average discharge of 1 kkg As/yr/plant EPA, 1979b, see Appendix A, Note 11. There are 5 plants discharging directly and one plant discharging indirectly. Atmospheric emissions based on 0.6 kg As emitted/kkg Zn processed (Davis, 1971) x 472,480 kkg Zn produced (Cammarota, 1980). This estimate is probably too high due to use of unsubstantiated emission factors. EPA, 1976b estimates that the emissions from all primary zinc smelters total 80 kkg/yr. This estimate, however, is also unsubstantiated. Water value includes 540 kkg As released to surface waters from lead/zinc mines, see Table 3-4.

Table 3-3. (Concluded)

i) Wastewater data based on 0.12 kkg As discharged/plant and 27 directly discharging plants, EPA 1976b. See Appendix A, Note 11.

j) Based on EPA, 1976a. See Appendix A, Note 14.

k) Input based on phosphate rock mine shipments (P_2O_5 content) and arsenic content of Florida rocks (15,728,287 kkg, As = 45 g/kkg) EPA 1976a; Stowasser, 1978. See Table A-4 and Appendix A, Note 17 for distribution calculations.

l) Based on EPA, 1976a, by analogy to phosphorus. See Appendix A, Note 13.

m) Based on 2,800 kg slag generated/kkg antimony produced, 3g As/kkg slag, and 210 kg solids in sludge/kkg antimony produced, containing 16g As/kkg. See Appendix A, Note 10, EPA, 1978a.

n) See Appendix A, Note 15.

Table 3-4. Inadvertent Releases of Arsenic from Mining and Milling, 1979 (kkg)

Source	Production (kkg)	Estimated Environmental Releases				
				Water		
		Air	Land	Surface	POTW	Total
Copper[a]	62,180,500 (concentrate)	110	630	14		754
Lead[b]	992,250	neg		550		550
Zinc						
Aluminum[c]	1,960	neg		180		180
Antimony[d]	250	neg			neg	neg
Coal[e]				94		94
Iron ore[f]	87,087,500 (crude ore)	3				3
TOTAL		113	630	830		1,581

a) Wastewater value based on As concentrations and flow rates for copper mines and mills EPA, 1975c. See Appendix A Note 12. Land discharge based on tailing pond concentrations, see Appendix A, Note 12. Atmospheric emissions based on 248,722,000 kkg ore mined (Butterman, 1980) x 0.45 x 10^6 kkg As emitted/kkg ore (Davis, 1971).

b) Wastewater discharge based on EPA 1978b average discharge rate of 0.044 kkg/day for 34 discharging lead/zinc mines operating 365 days per year. Atmospheric emissions based on 0.45 x 10^{-6} kkg As emitted/kkg ore produced and production of approximately 992,250 kkg lead and zinc = 0.49 kkg (EPA, 1979; Ryan *et al.* 1978; Cammarota 1978).

c) Based on estimated annual discharge rates for the 2 bauxite ore processing facilities of 0.26 kkg As /day x 350 day/yr EPA, 1979e. Atmospheric emissions based on 0.45 x 10^{-6} kkg/kkg ore produced (EPA, 1979b) and ore production of 1,960 kkg (Kurtz, 1978) = 0.001 kkg.

d) EPA 1975c. Wastewater discharge based on maximum flow from flotation mill of 342,990 l/day 350 d/year x 0.23 mg As/l = 0.02 kkg As. Atmospheric emissions based on 0.45 x 10^{-6} kkg As/kkg ore produced x 250 kkg ore produced = 0.0001 1 kkg (EPA, 1979e; Rathjen, 1978).

e) Based on EPA screening sampling data for coal mining. Mine drainage contains 12 mg As/l x 3,800 l/day/mine x 5,673 mines x 365 day/yr = 94 kkg As. EPA, 1979e.

f) EPA, 1973b; Peterson, 1980. Based on an atmospheric emission factor of 0.1 kg As emitted/kkg arsenic present in ore, average arsenic concentration of 400 mg/kg, and 1979 crude ore production of 87,087,500 kkg ore.

Table 3-5. Estimated Environmental Releases of Arsenic from Use, 1979 (kkg)

Use[a]	% of Total Quantity Used	Quantity[b] (kkg)	Contained in Product (kkg)	Estimated Environmental Releases				
				Air	Land	Water Surface	Water POTW	Total
Pesticides[c]	70%	10,250	10,250	1,500	8,100	720		10,000
Wood Preservative	20%	2,930	2,930	neg[d]	neg[d]	neg[e]		neg
Glass Manufacture	5%	730	720[f]	10[g]	neg[h]	neg[i]		10
Alloys[j] (copper and lead)	3%	440	440	neg				neg
Other (total)	2%	290		2	10		50	
Feed Additives/		220	218	2[k]				2
Veterinary Chemicals							<50	50
Electronics		60	60[l]					
Catalysts		10			10[l]			10
TOTAL	100%	14,640		1,500	8,100	720		10,311

a) Use distribution pattern based on percentages from Loebenstein, 1980a.

b) Quantity is kkg of As.

c) Includes herbicides, cotton dessicants, defoliants, and soil sterilizers. See Table 3.2

d) Assume all 476 plants generate sludge, sludge has 9 g As/kkg dry sludge, (EPA, 1979d), sludge density 1.03, wet sludge is 95% water and sludge generation of 0.016 yd^3/1,000ft^3 wood treated, 92,903,000 ft^3 wood treated/yr, (USDA, 1980), See Appendix A, Note 5.

e) Based on arsenic concentrations and flow rates from selected wood preserving facilities, EPA, 1979d. See Appendix A, Note 4.

f) About 70-99% of the arsenic used remains in the glass. Using 99%, about 720 kkg of As are contained in glass (EPA, 1980b).

g) Based on data from EPA, 1980b; 0.015 kg As emitted/kkg pressed and blown glass produced and a production of 634,900 kkg. See Appendix A, Note 3.

h) All off quality glass is recycled (EPA, 1980b). Negligible is defined as less than 1 kkg.

i) See text, Section 3.4.3.

j) EPA, 1976a, EPA, 1980b. Atmospheric emissions from nonferrous alloying are negligible; see Appendix A, Note 16.

k) Assume 75% of 290 kkg for "other" is consumed in feed additives, 25% in electronics. Based on 10 kg As released/kkg arsenic processed in feed additive manufacture, assuming manufacture of arsenical feed additives involves similar techniques to arsenical pesticide manufacture, EPA, 1976a. See text for veterinary chemical discharge to POTW.

l) All arsenic is contained in the product, i.e., light emitting diodes. Spent catalysts are assumed to be disposed to land. Assume ~90% (60 kkg) of arsenic used in electronics, 10% in catalysts

Table 3-6. Arsenic Releases from Production and Use of Pesticides, 1979 (kkg)

Pesticides	Quantity (kkg As)	Estimated Environmental Releases				
				Water		
		Air[a]	Land[b]	Surface	POTW	Total
MSMA[c]	3,000					
Production		2-65	-	neg[d]	neg[d]	65
Use		500[e]	2,200	210[b]		2,900
DSMA[c]	670					
Production		0.5-16		neg[d]	neg[d]	16
Use		110[e]	500	46[b]		650
Arsenic Acid	2,830					
Production		2-54		neg[f]	neg[f]	54
Use		17[g]	2,600	200[b]		2,800
Cacodylic Acid	690					
Production		0.4-13		neg[h]	neg[h]	13
Use		120[e]	510	48[b]		680
10,10' - OBPA	4					
Production				neg[i]		
Use						
Other[j]	3,060					
Production		2-60		neg[k]	neg[k]	60
Use		510[e]	2,300	214[b]		3,000
TOTAL[l]	10,250	1,500	8,100	720		10,000

Footnotes next page.

Table 3.6. (Concluded)

a) All atmospheric emissions ranges from pesticide manufacture (EPA, 1973b) are based on a worst case uncontrolled emission factor of 10 kg As omitted/kkg pesticide produced and a controlled value of 0.29 kg/kkg (EPA, 1980b). Actual emission value is probably nearer the lower end of range due to increase in air quality regulations.

b) All land discharges are by difference, i.e., 3,000 kkg As present in MSMA-65 kkg emitted to air from production - 500 kkg emitted to air from application - 210 to water as runoff = 2,435 kkg to land. Discharge to surface water based on 7% of the total quantity of arsenic aplied lost as run off (Richardson *et al.*, 1978); e.g., for MSMA: 3,000 kkg As applied x 0.7 =210 kkg.

c) Actual quantities produced are considered proprietary. EPA (Keany, 1980) estimates 8,160 to 11,800 kkg MSMA/DSMA produced per year of which 3,630 kkg are exported. Using the larger value (11,800-3,630 = 8,170) and assuming 20% of the toal production is DSMA (EPA, 1980b) 6,530 kkg are MSMA, 1,630 kkg DSMA. Based on arsenic content of 46% in MSMA and 41% in DSMA, 3,000 kkg and 670 kkg of arsenic are contained in MSMA and DSMA respectively.

d) EPA, 1975d. Based on 0.23 kg As discharged/day x 350 day/year operation at a representative MSMA/DSMA facility; there are currently 3 manufacturers (EPA, 1980b).

e) Emissions from use based on 168 kg As emitted/kkg As applied as pesticide, Davis and Associates, 1971.

f) EPA, 1975e. No wastewater associated with arsenic acid production; waters are all recycled.

g) Based on EPA, 1977 emission factors for ground application of arsenic acid of 6.1 kg As/kkg As used.

h) EPA, 1976a. Sittig, 1979; EPA, 1976c.

i) Negligible due to very small amount of arsenic used in production; all arsenic is assumed to be contained in end product. Quantity produced assumed to be similar to that produced in 1974 (EPA, 1976a).

j) Includes Paris green, lead arsenate, sodium arsenite, for which individual production data are confidential.

k) EPA, 1975e. No wastewater discharge is reported for production of calcium arsenate or Paris green. Lead arsenate manufacture at one facility generates no wastewater, however, some producers reportedly discharge a filtrate liquid. Concentrations of arsenic in this stream are not reported.

l) Totals may not add due to rounding.

Environmental releases of arsenic from production of MSMA and DSMA total 65 kkg and 16 kkg, respectively; essentially all of these releases are into the atmosphere. On the basis of annual discharge of 0.08 kkg arsenic at a representative DSMA/MSMA production facility, wastewater discharges are believed to be negligible (EPA 1975d, EPA 1980b).

Larger quantities of arsenic are released to the environment from pesticide application; approximately 500 kkg and 110 kkg of arsenic are emitted to the atmosphere from MSMA and DSMA use annually (see Table 3-6). Approximately 2,200 kkg and 500 kkg arsenic are disposed to land, from MSMA and DSMA use respectively, either during application or as fields are tilled. Discharge to water occurs as runoff; DSMA and MSMA are not intentionally released to surface waters or POTWs. Richardson et al. (1978) determined that for arsenic acid, about 7% of the amount of arsenic applied is lost as runoff and erosion. If this loss is similar for MSMA/DSMA, then 210 kkg and 46 kkg of arsenic would be discharged per year from MSMA and DSMA use, respectively.

3.4.1.2 Arsenic Acid

Arsenic acid (H_3AsO_4) is produced on a batch basis from arsenic trioxide and nitric acid (see Appendix B, Figure 2). Currently it is used as a cotton dessicant to facilitate mechanical harvesting (Richardson et al. 1978). In 1979, approximately 2,830 kkg of arsenic were contained in arsenic acid. Based on uncontrolled and controlled emission factors of 10 kg As/kkg pesticide produced and 0.29 kg As/kkg pesticide produced, respectively, 2 kkg to 54 kkg of arsenic were emitted to the atmosphere during arsenic acid production in 1979 (EPA 1973b; EPA 1980b).

Due to increases in application and efficiencies of emission control devices and additional state and federal regulations, the actual quantity of arsenic emitted is probably closer to the lower portion of the range, i.e., 10 kkg. Wastewaters from production are recycled to NO_x scrubbers or returned to the reaction vessel for further product recovery; therefore, no wastewater is discharged from this process (EPA 1975c).

During application of arsenic acid, approximately 17 kkg of arsenic are emitted to the atmosphere; 2,600 kkg are disposed of on land (Table 3-6). Discharge of arsenic to waters occurs as runoff. Richardson et. al. (1978) determined that 7% of the arsenic applied is lost as runoff; on this basis, in 1979 approximately 200 kkg of arsenic entered surface waters from use of arsenic acid as a cotton defoliant.

3.4.1.3 Cacodylic Acid

Cacodylic acid, an insecticide and contact herbicide, is manufactured by addition of calcium chloride and sulfur dioxide to DSMA (Sittig 1977). Its chief use is in lawn renovation and weed control in noncrop areas (EPA 1976d). A maximum of 13 kkg of arsenic is emitted to the atmosphere from production of cacodylic acid, based on EPA emission factors (Table 3-6).

Of the total 680 kkg of arsenic released to the environment from use of cacodylic acid, 17% (120 kkg) were emitted to the atmosphere, 76% (510 kkg) were disposed of on land, and 7% (48 kkg) were discharged to water as runoff (Table 3-6).

3.4.1.4 Miscellaneous Pesticides

Approximately 3,060 kkg of arsenic were contained in Paris green, lead arsenate, and sodium arsenite produced in 1979; production data for the individual pesticides are considered proprietary information. Production process diagrams for these pesticides are given in Appendix B. Environmental releases of arsenic from production of lead arsenate, Paris green, and sodium arsenite are chiefly atmospheric. Considering both stack and dispersive nonpoint emissions, 2 to 60 kkg of arsenic would be emitted annually from production (EPA 1973b, EPA 1980b) (see Table 3-6). Releases of arsenic from use of these pesticides totalled 3,000 kkg, of which 510 kkg were emitted to the atmosphere, 214 kkg were discharged to surface water, and 2,300 kkg were destined for land disposal (Table 3-6).

A small volume fungicide, whose chief component is 10, 10'-oxybisphenoxarsine (10,10'-OBPA), is used in plasticized polyvinyl chloride products. Total arsenic consumed in its manufacture, based on 1974 data, is 4 kkg (EPA 1976a); environmental releases of arsenic from production and use are thought to be negligible, due to the small quantities of arsenic consumed.

3.4.2 Wood Preservatives

Arsenical wood preservatives, chromated copper arsenate (CCA) and fluor chrome arsenate phenol (FCAP), are used for the most part to treat lumber, timber, fence posts, and cross ties. In 1979, approximately 2,930 kkg of arsenic were consumed in manufacture of wood preservatives. In 1978, 2,787,090 m^3 (92,903,000 ft^3) of wood products were treated with arsenicals (USDA 1980). Approximately 20% of all wood products treated with any type of preservative were preserved with arsenicals.

As noted in Table 3-5, releases of arsenic to the environment from production and use of CCA and FCAP are negligible. Appendix A, Note 4, outlines wastewater discharges. Based on an average discharge of 0.001 kkg per year per plant and a total of 476 plants, 0.5 kkg of arsenic are discharged annually (EPA 1979d). Sludge is generated from in-plant wastewater treatment; the arsenic content ranges from 6 g/kkg to 9 g/kkg dry sludge. Thus approximately 0.0005 kg arsenic would be sent to land via sludge disposal (see Appendix A, Note 5).

Arsenical wood-preserving compounds bind very tightly to wood fibers; release of arsenic from deterioration of treated wood is expected to occur very slowly, (EPA 1976a).

3.4.3 Glass Manufacture

In 1979, about 730 kkg of arsenic were utilized in glass manufacturing as a decolorizer, a fining agent (to remove bubbles), and as a stabilizer for color-producing chemicals (EPA 1980b). Quantities of arsenic used in these applications are decreasing as nonarsenical alternatives (various sulfates and antimony oxide) are found. Currently, opal and lead glass and certain specialty glasses are the largest consumers of arsenic (Sutherland 1980, Thatcher Glass Company 1980).

During glass manufacture, arsenic volatilizes and is thought to condense on particulates. Based on a controlled emission factor of 0.015 kg As emitted/kkg glass produced, and production of 534,900 kkg, about 10 kkg of arsenic are emitted to the atmosphere annually from glass manufacture (see Appendix A, Note 3). As all off-quality glass is recycled (as opposed to disposed of on land), release of arsenic to land is negligible (EPA 1980b). Arsenic is tightly bound in glass; therefore, release of mobile arsenic from discarded glass products sent to landfills is negligible. Based on EPA process descriptions and water use patterns (cooling water, washing of finished products), discharge of arsenic is negligible from production processes (EPA 1980b, EPA 1973a).

3.4.4 Alloys

Due to its semi-metallic properties, arsenic is added to lead, copper, and brass to enhance physical and chemical properties of those metals. Tables 3-4 and 3-5 outline environmental releases of arsenic from alloying (secondary lead/copper production). As recycling of arsenic-containing metals is so extensive, the environmental releases of arsenic contained in scrap metal are difficult to distinguish from releases of arsenic that is added during metal reprocessing. Therefore, these releases are calculated together, although about 440 kkg of "new" arsenic are added annually.

Atmospheric emissions from alloying are considered negligible (EPA 1976a, EPA 1980b). Land-destined arsenic (total of 300 kkg As) from these processes is contained in slag. Wastewater discharges are negligible (see Appendix A, Notes 7, 8, 11 and 19).

3.4.5 Small-Volume Uses of Arsenic

Approximately 290 kkg of arsenic (2% of the available arsenic supply) are consumed each year in feed additives, veterinary chemicals, electronics, and catalysts; releases of arsenic to the environment from manufacture and use of these products is presented in Table 3-5.

3.4.5.1 Feed Additives and Veterinary Chemicals

Arsenic is added to poultry and swine feed to prevent disease, and improve weight gain; roxarsone, carbarsone and nitarsone are among the most commonly used. Atmospheric emissions from manufacture of these compounds are thought to be similar to those of pesticides and total approximately 2 kkg arsenic. The remaining arsenic is assumed to be contained in animal excrement and disposed of on land. However, Goihl (1979) states that after anaerobic decomposition of animal wastes, arsenic levels decreased almost 40% from the level found in fresh waste. The loss is perhaps due to volatility of arsenic hydride metabolites.

Few data were available concerning veterinary chemicals. However, an EPA (1979g) study around a single veterinary pharmaceutical plant (which also produced organic arsenicals) indicates that wastewater discharges are probably less than 50 kkg/yr. The treated effluent discharged 50 g As/m^3 day; this stream was clarified, neutralized and settled before discharge to a POTW.

3.4.5.2 Electronics and Catalysts

Approximately 70 kkg of arsenic are used per year in light emitting diodes (LED) and catalysts. Specific quantities of arsenic used in LED and semiconductor manufacture are considered proprietary information; it is assumed that 90% (60 kkg) are used in electronics and 10 kkg are used as catalysts. Such an assumption seems reasonable in that demand for semi-conductors/LED is higher than that for arsenical catalysts (used as tertiary arsines in coordination compounds for ether formation and hydrogenation of alkenes; other metal-containing catalysts are more common).

Most of the arsenic utilized in semi-conductors (in the form of gallium arsenide phosphate, GaAsP) is contained in the product (Hollenbeck 1980). Since GaAsP is formed by vapor-phase reactions (Penz et al. 1979, Carapella 1978), release of arsine gas is possible. As the maximum allowable exposure limit for arsine gas is 0.05 ppm (TWA, 8 hrs), such emissions are most likely well controlled and probably total much less than 1 kkg per year. Such an estimate seems reasonable as even a worst case scenario of 1% loss would emit 0.6 kkg. The potential for occupational exposure through this route, however, does warrant further investigation.

Arsenic is used as a catalyst during hydrogenation of hydrocarbons, in paper manufacture, and ozonation of cyanides (EPA 1976d). No specific data were found; it is, therefore, assumed that all arsenic-containing spent catalysts are disposed of on land.

3.5 DISPOSAL OF ARSENIC-CONTAINING WASTES

This section addresses the ultimate disposition of arsenic discharged to municipal waste facilities: publicly-owned treatment works (POTWs) and urban refuse landfills or incinerators. A summary materials balance around each waste treatment category is shown in Table A-6 (Appendix A).

3.5.1 POTWs

Arsenic loading to POTWs is largely dependent upon variations in industrial discharges and the type of industry in a particular municipal area. A framework for calculating the total arsenic flow through the nation's POTWs (see Table A-7) is provided by data from a recent EPA study. A materials balance of arsenic at the treatment plants can be constructed using a total POTW flow of approximately 10^{11} l/day (EPA 1978c) and median values of <50 µg As/l (influent) and <50 µg As/l (effluent) (EPA 1980d). It is assumed for purposes of these calculations that influent and effluent flow rates are equal, i.e., that water loss from sludge removal and evaporation are small compared with influent flows. The results of the calculations show influent and effluent loadings to be the same, 1,800 kkg/yr. Trace metal removal can be achieved with advanced treatment processes; however, less than 2% of the nation's POTWs utilize such processes (Linstedt et al. 1971). Arsenic removals of 90% have been demonstrated using ferric chloride precipitation; combining that treatment with activated carbon yields removals of up to 97% (Reimers and England 1980). However, plants surveyed to obtain the previously described influent and effluent arsenic concentrations did not practice such techniques.

It is further assumed that while arsenic is recycled within the activated sludge process, all will eventually be wasted. Thus, the value for arsenic in sludge should be the difference between the influent and effluent arsenic totals, as there is an assumed negligible loss of arsenic to the air. Using this assumption, however, no arsenic would be released to sludge, an erroneous conclusion, as seen from the sludge data presented in Table A-7. This conflict can be attributed to mathematical assumptions made necessary by the imprecise nature of the data (e.g., high detection limits).

An alternative method, therefore, for estimating the annual arsenic discharge to sludge is to calculate arsenic release from the arsenic concentration and quantity of dry sludge produced annually (6×10^6 kkg, EPA 1979i). Assuming the median arsenic concentration of POTW wet sludge to be 170 µg/l (EPA 1980e) and that wet sludge is 95% water by weight, approximately 20 kkg are discharged to land. Approximately 25% of all municipal sludge is landfilled, 25% spread on land, 15% ocean dumped and 35% incinerated. As ocean dumping of sludge is mandated to cease by 1981 and assuming that more stringent air quality standards curb incinerator use (EPA 1979h), the arsenic contained in sludge is assumed to be discharged to land.

3.5.2 Urban Refuse

Urban refuse, divided into combustible and noncombustible fractions, is usually landfilled (87%), recycled (8%) or incinerated (5%) (Geswein 1980, Alvarez 1980). Arsenic flow through a municipal incinerator with a capacity of 920 kkg dry refuse per week is illustrated in Figure B-4, Appendix B. The only available arsenic data for incinerators is for loading to fly ash and suspended particulates (Greenberg et al. 1978). Assuming 10^7 kkg of solid waste are incinerated per year (EPA 1976e), an estimated 8.7 kkg of arsenic were released to the atmosphere (suspended particulates) and to land (flyash), each. The land figure excludes a factor for bottom ash, however, which is assumed to account for the remainder of the environmental loading to this medium, thus completing the materials balance around this process, (see Table A-6). Aqueous arsenic discharges from incineration were not available, but by analogy to other heavy metals, it is probably not a significant amount (Law and Gordon 1979).

The estimate of the total input of arsenic to incinerators is based on an arsenic concentration of 17 mg/kg in the combustible fraction of municipal solid waste (U.S. Dept. of Interior 1979). If this concentration is also assumed to apply to the noncombustible fraction, at a minimum, a total input to landfill of at least 3,000 kkg can be calculated, utilizing the fact that 87% of the 2 x 10^8 kkg of municipal solid waste generated annually is landfilled (Geswein 1980, Gordon 1978, see Table A-6). Landfill losses of arsenic are difficult to quantify due to the exceedingly large number of sources and types of material. Of the total amount of municipal solid waste produced annually, approximately 10% by weight is metal, with 1% being nonferrous metal (Gordon 1978). No information is available as to what percentage of this amount of nonferrous metal is arsenic.

3.5.3 Urban Runoff

Urban runoff usually contains arsenic at concentrations on the order of 50 µg/l (U.S. EPA 1979j). Potential sources of arsenic release in urban areas include atmospheric fallout from combustion processes and smelters and runoff from pesticide use and natural deposits. Assuming an annual urban runoff flow rate of 2.1 x 10^{13}l/year (EPA 1977b), then an annual loading rate of 1,050 kkg can be estimated (see Appendix A, Note 21). Approximately 431 kkg (41%) flows to separate storm sewers and 452 kkg (43%) to unsewered areas. The remaining 167 kkg flows to combined sewers, which discharge to POTWs (U.S. EPA 1977b).

3.5.4 Natural Loading

Arsenic is a constituent of an estimated 245 mineral species; weathering and erosion of continental rock is, therefore, a source of arsenic input into the environment. Based on the total quantity of material released to the oceans via weathering worldwide per year (9.3 x 10^9 kkg EPA 1980e), the percentage of that total that is igneous, shale, and limestone and their average arsenic content, approximately 2,850 kkg of arsenic are discharged to U.S. waters per year via erosion (see Appendix A, note 20). However, intensive land use would increase the total quantity of arsenic released about four times (EPA 1980e); as definitions of intensive and non-intensive land use are unclear, an average of the two estimates of arsenic discharge, 7,100 kkg/yr is probably the best estimate for natural loading.

Such an estimate seems reasonable in that the first estimate, 2,850 kkg/yr, is based on 1968 land use data; a larger estimate obtained by averaging intensive and "non-intensive" estimates would most probably reflect the current patterns of land use.

3.6 SUMMARY

Arsenic is recovered domestically as a byproduct of copper smelting. The only arsenic production facility, located in Tacoma, Washington, produced 280 kkg arsenic "metal" and arsenic trioxide containing 6,420 kkg As in 1979. Approximately 8,940 kkg arsenic were imported in 1979 and 1,000 kkg exported. Table 3-1 and Figure 3-1 outline environmental releases from production, use, and inadvertent sources of arsenic. Of the total 1,400 kkg arsenic lost ot the environment during production, 85% (1,200 kkg) was land disposed, 15% (210 kkg) was emitted to the atmosphere, and <1 kkg was discharged to surface waters. Furthermore, the 1,400 kkg As released to the environment from its production account for only 3% of the total arsenic released from all sources (i.e., production, use and inadvertent sources; see Table 3-1).

The pesticide industry is by far the largest consumer of arsenic; production of arsenical pesticides utilized approximately 10,000 kkg (70% of the available supply) arsenic in 1979. Of the total 10,000 kkg As released to the environment from production and use of pesticides, 1,500 kkg (15%) were emitted to the atmosphere, 8,100 kkg (81%) were land-disposed and the remaining 720 kkg (~5%) were discharged to surface waters. Production and use of wood preservatives, glass and alloy additives and miscellaneous arsenicals, consuming 30% of the available U.S. supply, released approximately 70 kkg arsenic to the environment. The total 10,070 kkg As released to the environment from all uses of arsenic represents approximately 20% of the total 51,000 kkg released from all sources (production, use and inadvertent sources).

Arsenic is associated with most mineral ore; processing of these ores, as well as smelting and refining of the resultant metal, introduced 21,500 kkg of arsenic (42% of the total 53,400 kkg As released) into the environment. These releases are primarily contained in land-disposed slag. Fossil fuel combustion released 17,000 kkg (33% of the total quantity released from all sources) of arsenic to the environment; cotton ginning accounted for about 880 kkg.

Demand for arsenic is expected to increase only about 1% annually through 1985. This low market growth factor can be attributed to the search for non-arsenical alternatives, especially in the glass manufacturing industry. According to the industries involved, non-arsenical wood preservatives, feed additives, and cotton dessicants are not as effective or economically attractive as are arsenic products.

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4.0 FATE AND DISTRIBUTION IN THE ENVIRONMENT

4.1 INTRODUCTION

This chapter describes the fate and distribution of arsenic in soil, water, and air following its environmental release from the processes described in Chapter 3.0. Emphasis is placed on the pathways leading to arsenic contamination of surface and ground water and potential exposure of humans and aquatic life. To the extent possible, the chemical form of arsenic initially released is identified for each major category of discharge and its transformations and transport to other media are followed to a point of equilibrium. This type of analysis is difficult and at times imprecise due to the multiplicity of arsenic forms released to the environment, as well as the numerous forms present naturally in soil and water. Due to the poor analytical techniques available for many forms of arsenic and the complexity of multi-variable analysis, many fate studies do not distinguish between different forms of arsenic. Therefore, it is not possible to maintain a mass balance for each chemical form from release (or formation) to exposure of receptors. At best, predominant forms in environmental media present at the time of exposure are identified.

The environmental fate and distribution of arsenic is analyzed by the characterization of specific environmental pathways. Once arsenic is released to air or water, the initial chemical form has less influence on the ultimate fate than the environmental parameters at a specific locale. In addition, since arsenic in certain forms is relatively mobile compared with other elements, the initial receiving medium often will not be the same one to which human and other receptors may be exposed. Therefore, elucidating the environmental fate of arsenic requires characterizing the major types of releases into environmental media and following each pathway through the various transformations and removal processes to the ultimate fate of the element.

The major pathways of physical transport of arsenic are designated in Figure 4-1 for the major categories of releases identified in Chapter 3.0. Atmospheric releases (Pathway 1) consist primarily of point sources such as power plants and smelter stack emissions. Inadvertent releases of arsenic from combustion processes are among the largest man-made sources of arsenic into the atmosphere. Atmospheric releases often lead to highly localized pollution of air, soil and water in the vicinity of some sources, such as nonferrous smelters.

Pathway 2 considers arsenic in solid waste resulting from flv ash disposal, mining activity, smelting/refining activities, and municipal refuse. Presently, this pathway accounts for most arsenic releases, and as air and water pollution standards become more stringent, the amount of arsenic disposed upon land can be expected to increase.

Pathway 3 identifies arsenic released in industrial aqueous effluents, such as those from wood preservers and pesticide manufacturers. Most effluents are discharged to local surface waters; only veterinary chemical plants and several metals production facilities are known to discharge to publicly owned treatment works (POTWs). The fate of arsenic in POTWs and in industrial wastewater treatment is discussed in Pathway 4.

Pathway 5 considers intentional uses of arsenic in pesticidal applications agriculturally, to lawns and surface water--or as a contaminant of phosphate fertilizers.

Figure 4-2 provides a composite overview of the major environmental pathways of arsenic indicating the approximate contributions made by the major industries to air, soil, and water loadings based on estimated releases from Chapter 3.0. As indicated, the major portion of the environmental releases of arsenic are to land.

This chapter is organized into four sections. First (in Section 4.2) the general chemical, physical, and biological processes are described that affect the behavior of arsenic in surface water and soil systems. Then Section 4.3 discusses for each of the five major pathways the interplay of these fate processes as they determine the ultimate distribution of arsenic in environmental media. Field studies and selected, relevant, monitoring data are used to illustrate each pathway. A general discussion of concentrations detected in environmental media, from large-scale national surveys and some local studies, is presented in Section 4.4. Finally, Section 5.5 provides an overview of the environmental pathways of greatest significance for exposure of humans and other biota.

4.2 IMPORTANT FATE PROCESSES

4.2.1 Physiochemical Fate Processes

4.2.1.1 General Fate Discussion

Arsenic is usually referred to as metalloid and is chemically similar to phosphorus, which occurs above it in the Periodic Table. It is usually found associated in nature with sulphide ores in minerals such as arsenopyrite (FeAsS), niccolite (NiAsS), cobaltite (CoAsS) and tennantite ($Cu_{12}As_4S_{13}$). Weathering (by water, chemical reactions, wind, etc.) of arsenic-containing bedrock, coals and ores leads to release and mixing of these minerals into soil, water and the atmosphere. In addition, intentional releases are responsible for a large portion of the arsenic entering the environment annually.

A wide variety of chemical forms of arsenic, both inorganic and organic, are released and also exist in each type of ecosystem receiving the releases. The forms of interest with regard to environmental exposure and risk of humans and other organisms and the interrelationships between forms are discussed in the following section. Table 4-1 lists the environmentally significant forms of arsenic that are discussed. The general environmental chemistry of arsenic in aquatic ecosystems (fresh,

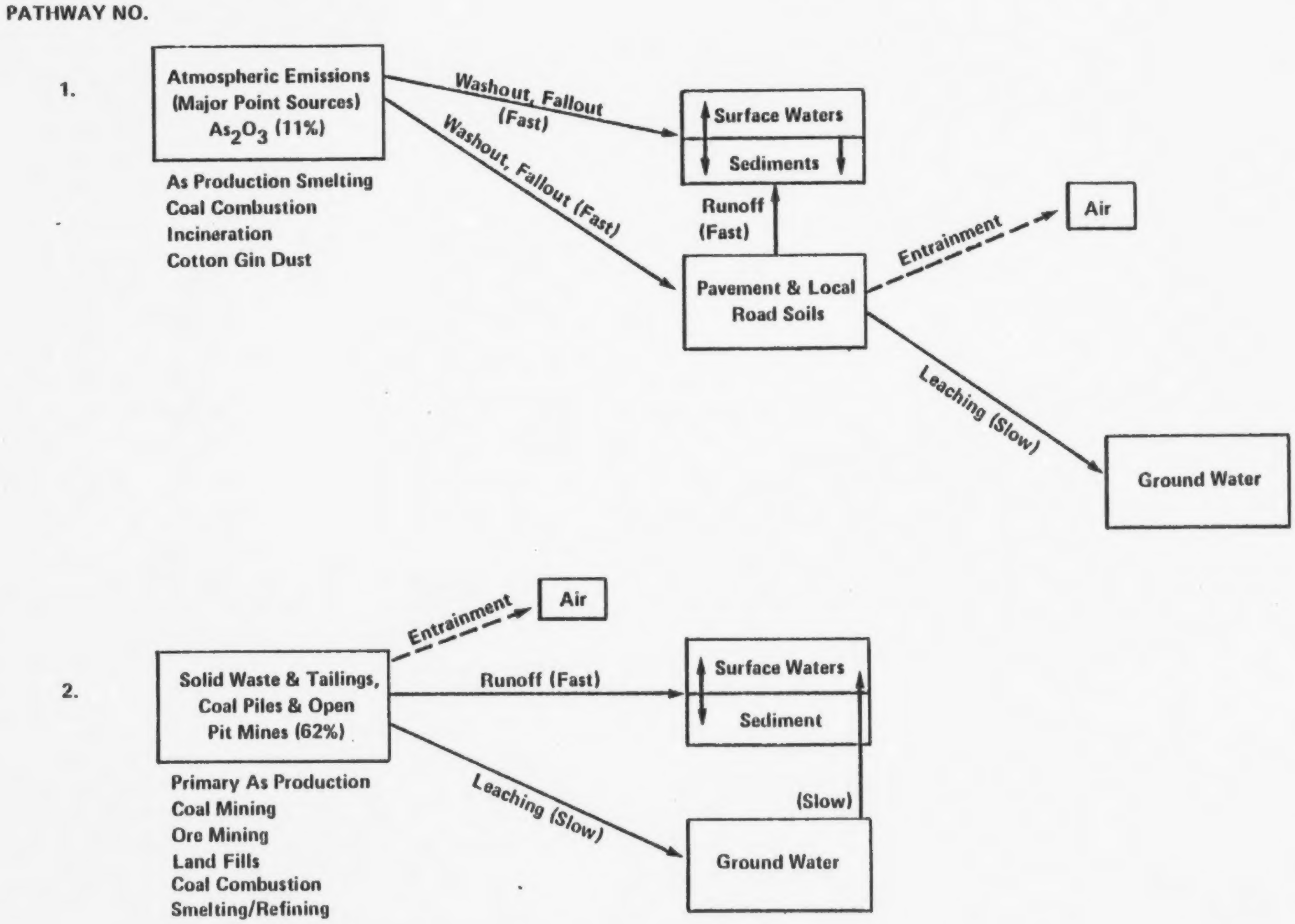

PATHWAY NO.
1.
Atmospheric Emissions (Major Point Sources) As_2O_3 (11%)
As Production Smelting
Coal Combustion
Incineration
Cotton Gin Dust
Washout, Fallout (Fast)
Washout, Fallout (Fast)
Surface Waters
Sediments
Runoff (Fast)
Pavement & Local Road Soils
Entrainment
Air
Leaching (Slow)
Ground Water
2.
Solid Waste & Tailings, Coal Piles & Open Pit Mines (62%)
Primary As Production
Coal Mining
Ore Mining
Land Fills
Coal Combustion
Smelting/Refining
Entrainment
Air
Runoff (Fast)
Surface Waters
Sediment
Leaching (Slow)
(Slow)
Ground Water

FIGURE 4–I MAJOR ENVIRONMENTAL PATHWAYS OF ARSENIC RELEASES

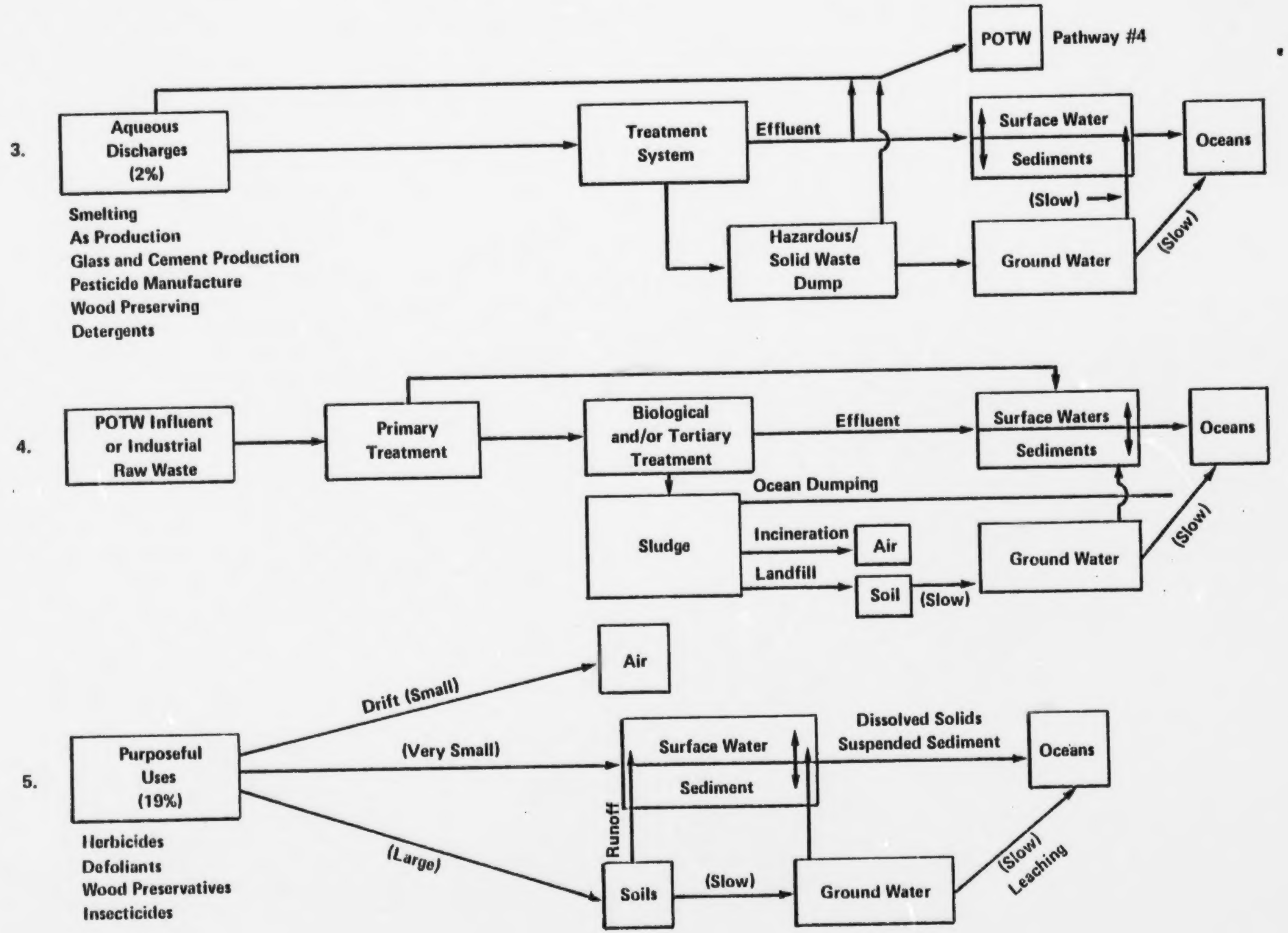
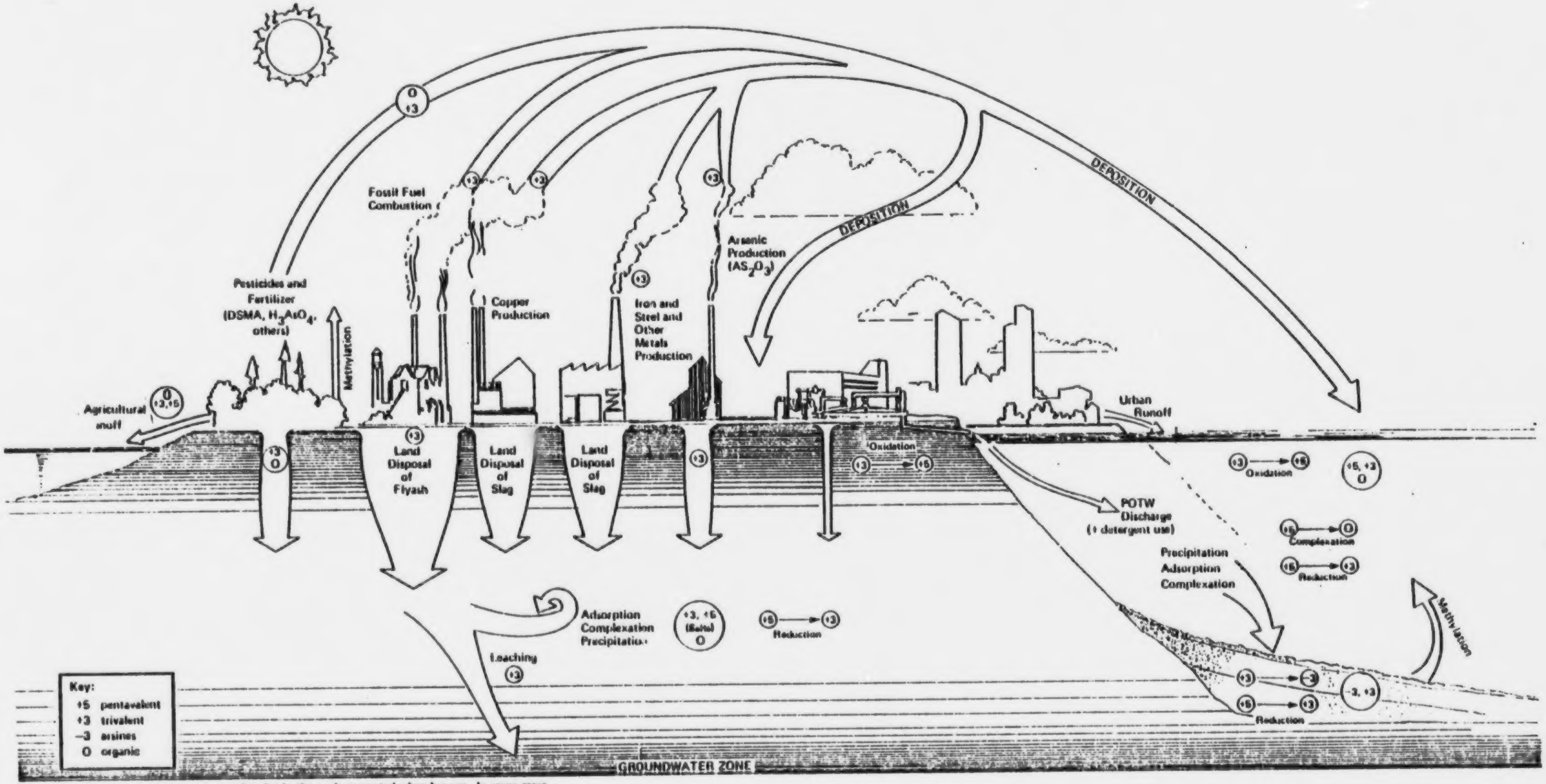

3.
Aqueous Discharges (2%)
Smelting
As Production
Glass and Cement Production
Pesticide Manufacture
Wood Preserving
Detergents
Treatment System
Effluent
POTW
Pathway #4
Surface Water
Sediments
Oceans
(Slow)
Hazardous/ Solid Waste Dump
Ground Water
(Slow)
4.
POTW Influent or Industrial Raw Waste
Primary Treatment
Biological and/or Tertiary Treatment
Effluent
Surface Waters
Sediments
Oceans
Ocean Dumping
Sludge
Incineration
Air
Landfill
Soil
(Slow)
Ground Water
(Slow)
5.
Purposeful Uses (19%)
Herbicides
Defoliants
Wood Preservatives
Insecticides
Drift (Small)
Air
(Very Small)
Surface Water
Sediment
Dissolved Solids
Suspended Sediment
Oceans
Runoff
(Large)
Soils
(Slow)
Ground Water
(Slow)
Leaching

FIGURE 4–1 MAJOR ENVIRONMENTAL PATHWAYS OF ARSENIC RELEASES (Continued)


0
+3
Fossil Fuel Combustion
+3
+3
+3
+3
DEPOSITION
DEPOSITION
Arsenic Production (AS_2O_3)
Pesticides and Fertilizer (DSMA, H_3AsO_4, others)
Methylation
Copper Production
Iron and Steel and Other Metals Production
Agricultural runoff
0
+3,+5
+3
0
+3
Land Disposal of Flyash
Land Disposal of Slag
Land Disposal of Slag
+3
Oxidation
+3 → +5
Urban Runoff
+3 → +5
Oxidation
+5, +3
0
POTW Discharge
(+ detergent use)
+5 → 0
Complexation
+5 → +3
Reduction
Precipitation
Adsorption
Complexation
Methylation
Adsorption
Complexation
Precipitation
+3, +5
(Salts)
0
+5 → +3
Reduction
Leaching
+3
+3 → -3
+5 → +3
Reduction
-3, +3
Key:
+5 pentavalent
+3 trivalent
-3 arsines
0 organic
GROUNDWATER ZONE

Note : The forms of arsenic present at each point in the cycle are noted when known. In some cases forms are unknown or other forms may also be present.

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FIGURE 4–2 MAJOR ENVIRONMENTAL PATHWAYS OF ARSENIC RELEASES

TABLE 4-1. ARSENIC FORMS OF ENVIRONMENTAL SIGNIFICANCE

Form	Examples	Comment
INORGANIC		
Arsenic trioxide	As_2O_3	Formed during combustion
Arsenous acids	$H_3AsO_3, H_2AsO_3^-, HAsO_3^{2-}$	Hydrolysis products of As_4O_4
Arsenic acid	$H_3AsO_4, H_2AsO_4^-, HAsO_4^{2-}$	Formed when As_4O_6dissolved in nitric acid
Arsenites	$\overset{*}{M}H_2AsO_3, M_2HAsO_3, M_3AsO_3$	Salts of arsenous acid
Arsenates	$MHAsO_4, M_3(AsO_4)_2$	Salts of arsenic acid
Arsenic sulfides	$AsS_2^-, HAsS_2$	Reaction product of hydrogen sulfide in presence of hydrochloric acid with trivalent or pentavalent arsenic compounds
ORGANIC		
Methanearsonic acid	$CH_3AsO_3H_2$	Methylation product of various arsenic compounds
Dimethylarsinic acid (cacodylic acid)	$(CH_3)_2AsOOH$	Methylation product of various arsenic compounds
Arsine	H_3As	Reduction and methylation products of inorganic and methylarsenic acids
Dialkylarsines	$HAs^{-3}\ (CH_3)_2$	Reduction and methylation products of inorganic and methylarsenic acids
Trialkylarsine (e.g., trimethylarsine)	$As(CH_3)_3$	Reduction and methylation products of inorganic and methylarsenic acids

* M signifies a univalent metal cation or an equivalent of a multivalent cation.

Adapted from Braman (1972), NAS (1977).

sediment, estuarine) and in soil (including ground water) is presented in 4.2.1.2 through 4.2.1.4. The behavior of arsenic in the atmosphere is not considered due to the emphasis placed on waterborne routes of exposure and risk in this report. (Atmospheric fate is only considered as a first step in fate pathways eventually leading to soil/ground water or surface water, and this discussion is presented in Pathway 1.)

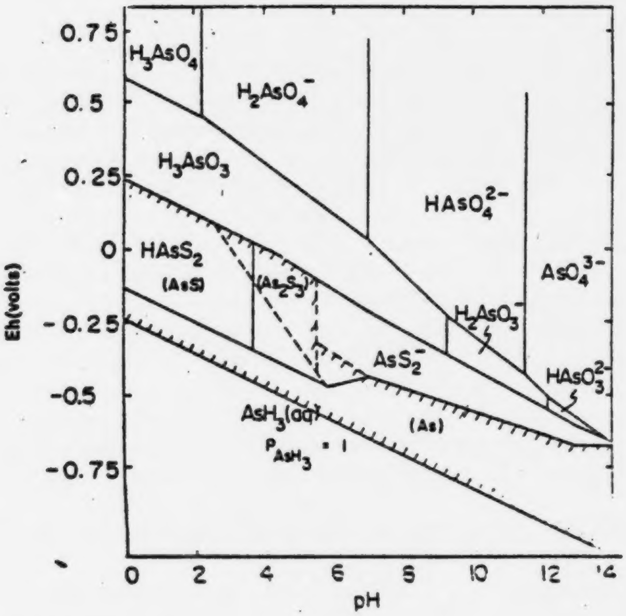
4.2.1.2 Aqueous Chemistry

In aqueous systems, arsenic is stable in four oxidation states, +5, +3, 0 and -3. An Eh-pH diagram (Figure 4-3) was constructed by Ferguson and Gavis (1972) for a simplified system containing only sulfur and arsenic.

An Eh-pH diagram provides a schematic description of the changes in arsenic speciation as a function of Eh (which signifies redox potential) and pH. Such a description can only be made for very simplified systems, far simpler than environmental conditions. However, it gives a rough idea of the predominant forms of arsenic existing at equilibrium in solution under a set of different conditions as determined by two influential environmental parameters, Eh and pH. In most aquatic systems, pH values range from 3 to 9. High Eh values are representative of aerobic waters and low Eh values of reduced environments low in dissolved oxygen. Figure 4-3 shows that in oxidized waters the arsenic acid species (H_3AsO_4) predominates below pH 2, with $H_2AsO_4^-$, and $HAsO_4^{2-}$ within the pH range of 2 to 11. In mildly reducing waters such as may be found in the hypolimnion of a lake, the arsenious acid species (H_3AsO_3) is the primary group, with $HAsS_2$ (aq) species appearing as the system becomes more reducing. Environments that are highly anoxic may contain arsine (AsH) and arsenic metal.

Dissolved arsenate (AsO_4) in oxidized waters may be removed by several mechanisms: adsorption onto iron and aluminum hydrous oxides and clays, coprecipitation with iron oxides, and isomorphic substitution with phosphorus in minerals. Most iron oxides are positively charged ionic species; arsenate is adsorbed as a negatively charged ion. Similarly, arsenite (AsO_3) is removed by the same processes.

Wagemann (1978) presented an Eh-pH diagram for a model system similar to that of Ferguson and Gavis (see Figure 4-3) except for the addition of a precipitant, barium. The main difference between the two model systems was that in oxidized waters above pH 4, the presence of barium caused precipitation of $Ba_3\ (AsO_4)_2$, which was a stable species. Wagemann suggested that copper, iron and chromium would play a similar role to that of barium. At lower pH levels in oxidized waters, the arsenious acid species predominated and in reduced waters, arsenious acid (H_3AsO_3) was stable over the pH range of 0 to 9. These results were compatible with those in the barium-free system of Ferguson and Gavis.



Source: Ferguson and Gavis (1972).

FIGURE 4–3 THE Eh-pH DIAGRAM FOR As IN AN AQUEOUS SYSTEM WITH ARSENIC AND SULFUR. SOLID SPECIES ARE ENCLOSED IN PARENTHESES IN CROSS-HATCHED AREA, WHICH INDICATES LOW SOLUBILITY.

Arsenic also undergoes biological transformations including methylation, oxidation and reduction. These reactions are discussed in detail in 4.2.2.

Ferguson and Gavis provide a mechanism for the cycling of arsenic in a typically stratified lake. Figure 4-4 illustrates arsenic species ($HAsO_4$) in the epilimnion that coprecipitate with iron oxides, sink down to the hypolimnion where reduction to $HAsO_2$ and AsS_2 occur, at the proper Eh and pH. In addition, coprecipitation or adsorption will bring arsenic to the sediments, where reduction of the iron to Fe (II) results in solubilization of the arsenic (and thus its return to the water column) or stabilization as a precipitated sulphide. Solubilization in the sediments may also occur by methylation and reduction of arsenic to arsine, which can volatilize from the aquatic system.

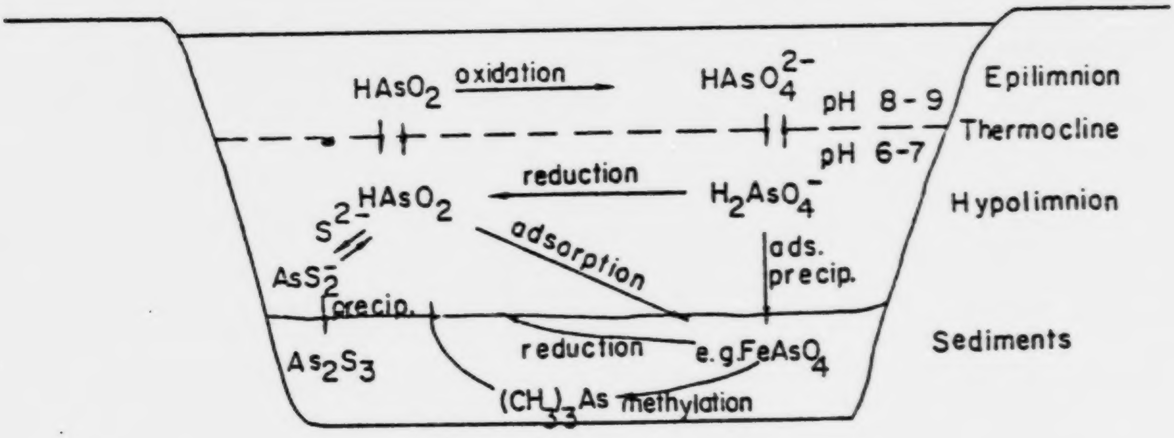
4.2.1.3 Sediment

Sediment in fresh and ocean waters is the recipient of arsenic compounds that precipitate with or adsorb onto clays, carbonates, and hydroxides. Arsenite (III) strongly adsorbs to metal sulphides. Holm et al. (1979) found in an arsenic-stressed river that the pore water of the sediment cores contained primarily inorganic arsenic, although methylated arsenic was the primary species in the ground water of the area. Most of the arsenic in the sediment core solids was contained in the iron and aluminum hydroxide fraction, followed by arsenic in the amorphous, exchangeable, and calcium (arsenoapatite) fractions. The sediment surface layer was the only part of the core in which $Fe(OH)_3$ was stable (due to the Eh) and this was the predominant form. A study by Huang and Liaw (1978) of arsenic fractionation in lake sediments confirmed this distribution; for a total As concentration of 4.9 µg/g, 39% was associated with iron and aluminum oxides, 16% with carbonates, 26% with apatites and silicates, and 6% with organic matter.

In rivers, arsenic is transported both as a dissolved solid and adsorbed to suspended matter. The adsorbed fraction can be quite significant as indicated by Trefry and Presley's (1976) finding that 70% of the total arsenic flux down the Mississippi River into the Gulf of Mexico is associated with particulates; 30% was estimated to be in dissolved form.

4.2.1.4 Soil Chemistry

An excellent review of the soil chemistry of arsenic is provided by Walsh et al. (1977). The chemistry is similar to that of phosphorus, especially in aerobic systems, where the behavior of arsenate closely resembles that of orthophosphate. Differences to be noted are arsenic's lability, oxidation states, and low accumulation in organic matter. In slightly reduced soil (e.g., temporarily flooded soils) arsenite (H_3AsO_3) species form, and in very reduced soils (e.g., bogs, swamps) arsine, methylated arsenic and elemental arsenic predominate. Microorganisms



$HAsO_2$
oxidation
$HAsO_4^{2-}$
pH 8 - 9
pH 6-7
Epilimnion
Thermocline
Hypolimnion
reduction
S^{2-}
$HAsO_2$
$H_2AsO_4^-$
AsS_2^-
adsorption
ads. precip.
precip.
reduction
e.g. $FeAsO_4$
As_2S_3
$(CH_3)_3As$
methylation
Sediments

Source: Ferguson and Gavis (1972).

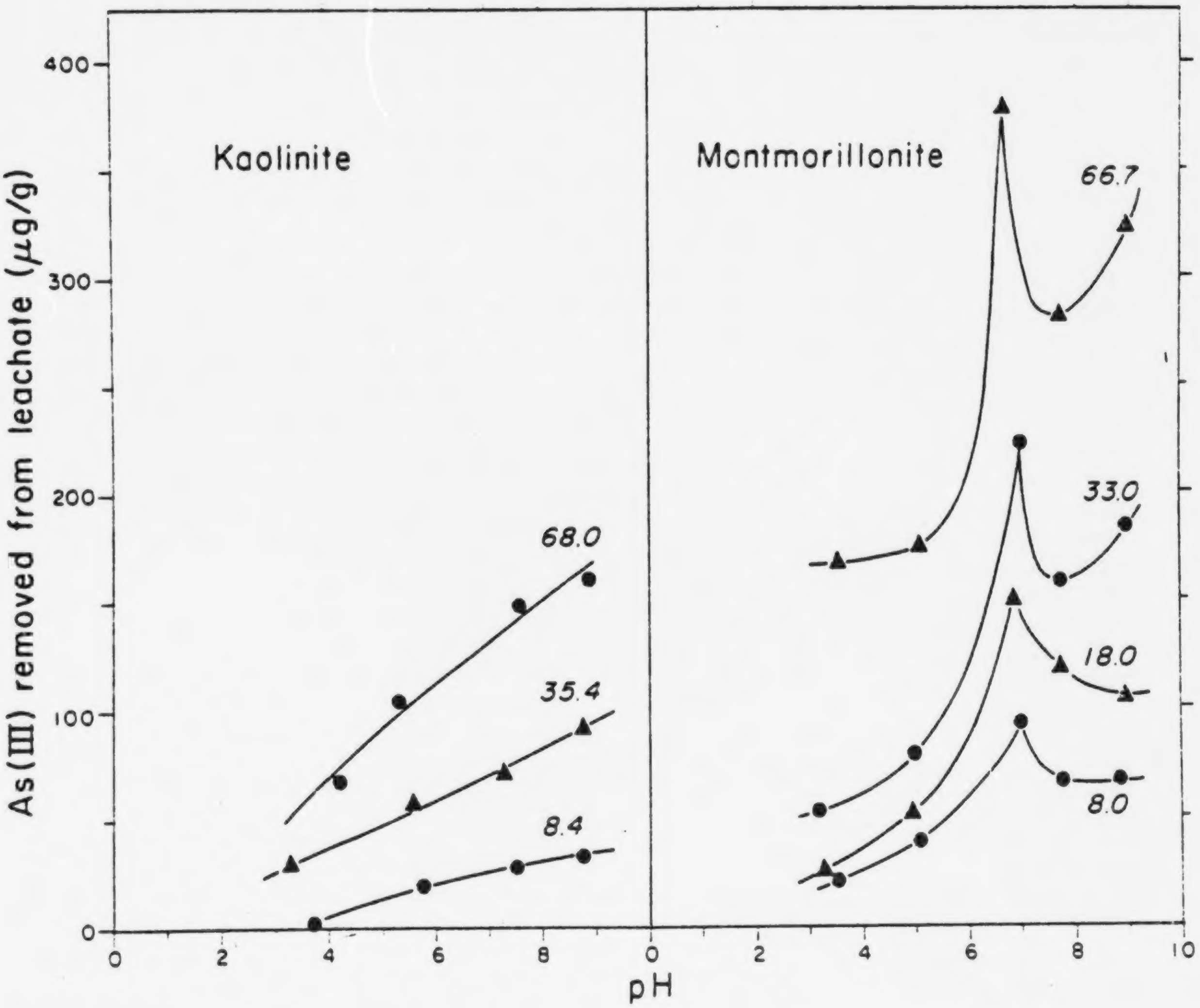
FIGURE 4—4 LOCAL CYCLE OF ARSENIC IN A STRATIFIED LAKE

are capable of oxidizing arsenite to arsenate in a matter of days in well oxidized soils (see following section on biological transformations). The principal adsorption mechanism for arsenic is anion exchange of the monovalent species (Griffin and Shimp 1978). Adsorption sites for arsenic species as in sediment, are provided chiefly by iron and aluminum hydrous oxides and clays. In a survey of U.S. soils, Woolson et al. (1971) found soil with a high reactive iron content to have arsenic predominantly in ferrous oxide complexes; soil with low iron content had arsenic either forming complexes with reactive aluminum or precipitating with exchangeable calcium. Figures 4-5 and 4-6 illustrate the adsorption of arsenic III and V on montmorillonite and kaolinite clays as a function of pH. Alkaline conditions are supportive of greater mobility. Application of phosphorus to soils contaminated with arsenic can possibly release adsorbed arsenic when phosphate replaces the arsenic on the adsorption site.

Walsh and Keeny (1975) estimated that a 3- to 6-month period is required before an equilibrium is reached for arsenic adsorption in most soils. The mobility of arsenic in ground water is in part determined by the redox potential, pH, and material components of the aquifer according to oxidation states: the oxidizing recharge area, a neutral transition zone, and the reducing discharge area. Sand column elution results indicated that As(III) is much more mobile than As(V) in ground water, regardless of the redox potential. As(V) eluted more quickly in reducing water than in oxidized water. The pH of the ground water also affects the amount of arsenic adsorbed to clay particulates and ferric hydroxides. As(V) is maximally adsorbed at low pH values (4-5); As(III) shows maximum adsorption at extreme pH values of >8 and <3. In light of these results, Gulens and Champ speculated that the increased mobility of As in reducing environments is due to (1) reduction of Fe(III) to Fe(II), releasing bound As(V); (2) reduction of As(V) to As(III); and (3) increased solution pH in the reduced waters.

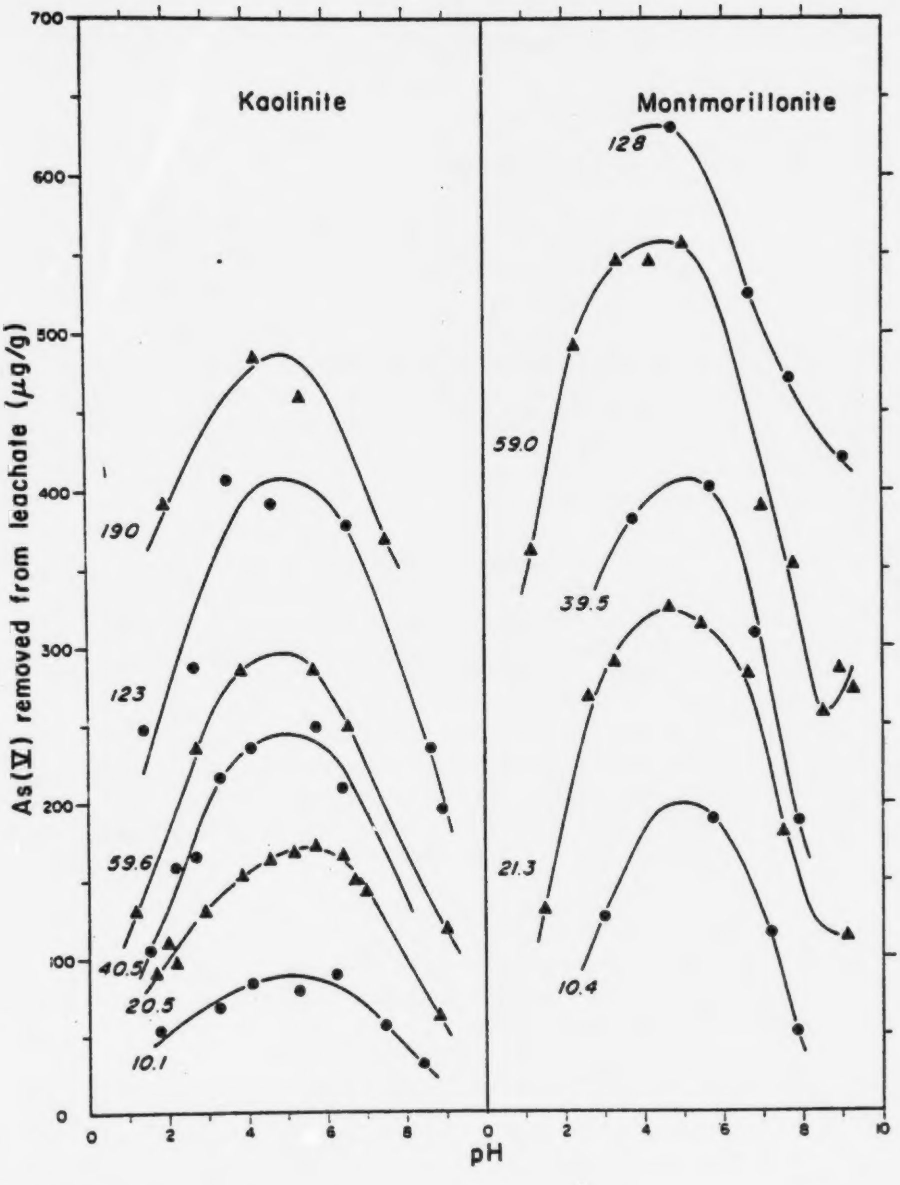
Griffin and Shimp (1978) studied the effect of pH and ionic competition of arsenate and arsenite adsorption from landfill leachate by kaolinite and montmorillonite clays. Montmorillonite was able to adsorb twice as much As as kaolinite clay. With regard to pH, the maximum adsorption of As(V) occurs at pH 5. The pentavalent form being adsorbed was thought to be H_2AsO_4. As(III) is increasingly removed by adsorption as the pH increases from 3 to 9. It is not known which As(III) species is being adsorbed. Relative to other heavy metals, both arsenic species are fairly mobile. Griffin and Shimp found that at pH 5, metal mobility increased as follows: Cr(III) < Cu < Pb < Zn < Ca < As(V) < As(III) < Se < Cr(VI).

Fuller (1978) examined the retention of arsenic in different soil types. In clay soils, arsenic has a low to moderate mobility, while in loams and sandy soils, the mobility is high with a velocity of 6-10 cm/day in loamy sand. The mass of arsenic adsorbed in soils was found to be correlated with clay content (r=0.88), surface area (r=0.66) and hydrous ion oxide (r=0.60). Less important parameters are pH, cation



Source: Woolson *et al.* (1977)

FIGURE 4-5 REMOVAL OF AS (III) FROM LEACHATE SOLUTIONS BY KAOLINITE AND MONTMORILLONITE CLAYS AS A FUNCTION OF pH



Source: Woolson *et al.* (1977)

FIGURE 4–6 REMOVAL OF AS(V) FROM LEACHATE SOLUTIONS BY KAOLINITE AND MONTMORILLONITE CLAYS AS A FUNCTION OF pH

exchange capacity (CEC), and manganese oxides. Leachate concentrations were as much as 73 mg/l As from a municipal landfill. In an earlier paper, Fuller (1977) had determined that the relative mobility of arsenic compared with other metals in neutral to alkaline soils (Griffin and Shimp studied acid soils) decreased as follows: Cr > As > Se > Cd > Be > Cu.

4.2.2 Biological Fate Processes

4.2.2.1 Microbial Biotransformations

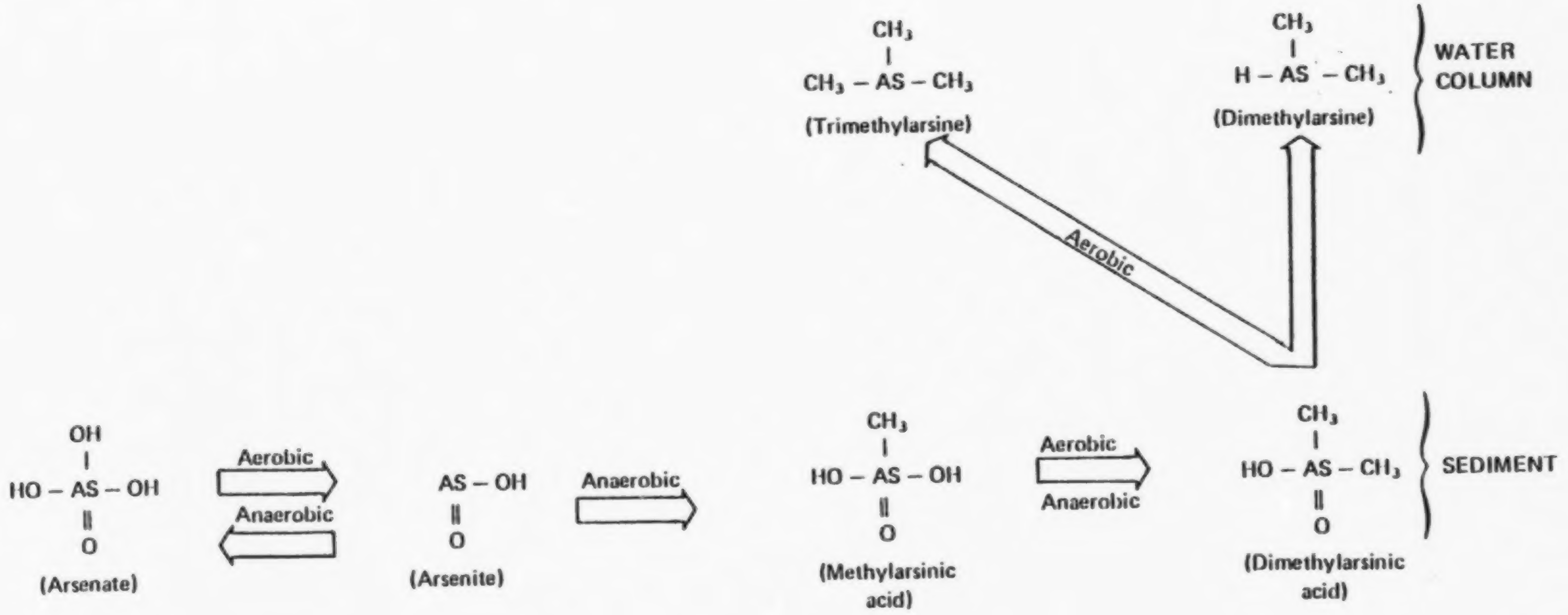
The speciation of arsenic in natural waters is significantly influenced by biota (Andreae 1978). Methylation is especially important in the transfer of arsenic from sediment back to the water column. Figure 4-7 illustrates this process.

Fungi, yeasts, algae and bacteria species have been reported to perform the various steps comprising the process. The function of methylation is thought to be detoxification for those organisms (Braman and Foreback 1973); however, it may increase mobility and result in exposure in other areas. The cycle occurs both in freshwater systems (Ferguson and Gavis 1972) and in saltwater systems (Andreae 1978).

Depending upon the environmental conditions (aerated or reduced, pH, microbial population), some or all of the steps in the overall cycle will take place. For example, in buffered, biologically active freshwaters, the rate of conversion of arsenite to arsenate via microbial oxidation may be significant (Ferguson and Gavis 1972). In soil, microbial transformation is thought to contribute appreciably to loss of applied arsenic from soil, up to 50% in one year (Pax 1973); however, the significant and persistent As residues reported in some soils suggest this is not always the case. Unfortunately, since few of these processes have been quantified and none under natural conditions, it is difficult to discern the role of microorganisms in the equilibrium speciation of arsenic.

Table 4-2 presents the results of a number of laboratory studies investigating biological transformations of arsenic. Most studies concerned pure cultures of microorganisms and were conducted in order to identify transformation products.

Methylation of both arsenate (via initial reduction to arsenite) and arsenite has been reported under aerobic and anaerobic conditions (McBride and Wolfe 1971, Andreae and Klumpp 1979, NAS 1977). The mechanism involves replacement of substituent oxygen atoms by methyl groups (Challenger 1947). Mono- and dimethyl compounds are common transformation products. Woolson and Kearney (1973) found dimethyl arsine to be the predominant product from reaction of cacodylic acid in soil (aerobic and anaerobic). It was unstable, oxidizing in air to the oxide or back to cacodylic acid. Under acidic conditions, sewage fungi produced trimethylarsine in sludge (Cox and Alexander 1973) and 70% of arsenite was transformed to trimethylarsine by fungi (Challenger 1947).


CH₃ – AS – CH₃ (with CH₃ above AS)
(Trimethylarsine)
H – AS – CH₃ (with CH₃ above AS)
(Dimethylarsine)
WATER COLUMN
Aerobic
HO – AS – OH (with OH above, O below)
(Arsenate)
Aerobic
Anaerobic
AS – OH (with O below)
(Arsenite)
Anaerobic
HO – AS – OH (with CH₃ above, O below)
(Methylarsinic acid)
Aerobic
Anaerobic
HO – AS – CH₃ (with CH₃ above, O below)
(Dimethylarsinic acid)
SEDIMENT

Source: Wood (1973).

FIGURE 4–7 ARSENIC BIOLOGICAL CYCLE IN WATER

TABLE 4-2. MICROBIAL TRANSFORMATIONS OF ARSENIC COMPOUNDS

Reaction	Species	Metabolic Product	Reference
Reduction and methylation of arsenate	*Methanobacterium*	Dimethylarsine	McBridge and Wolfe (1971)
Methylation of sodium cacodylate, sodium methanaersonate, sodium arsenite	Various fungi: *Pencillium aspergillus*, *Fusarium* sp., *Candida* sp., *Gliocladium* sp.	Trimethylarsine	NAS (1977)
Dimethylation of MSMA	Soil population	Arsenates and CO_2	NAS (1977)
Reduction of arsenate	Seawater bacterial population	Arsenite	Johnson (1972)
Methylation, reduction and complexing of arsenate	Marine algae	Arsenite, methylarsonate, dimethyl arsinate, and 12 soluble organo-arsenic compounds	Andreae and Klump (1979)
Methylation of arsenate	Sewage sludge population from anaerobic system	Assumed to be dimethylarsine (transformation complete in 8 hours)	McBridge, *et al*. (1978)
Methylation of arsenate	Fungi on bread cultures	Trimethylarsine (70% of As_2O_3 during 24 months)	Challenger (1977)

Reduction of arsenate and oxidation of arsenite have been reported in pure cultures of Chlorella, Micrococcus and Pichia and in mixed populations of pseudomonads and soil organisms (Alexander 1974). In estuaries, arsenic is thought to be relatively unreactive (Waslenchuk and Windom 1978), existing predominantly in the pentavalent form. In freshwater, monitoring data indicate the presence of arsenate, arsenite and methylated forms, with arsenate predominating (Braman and Foreback 1973).

Arsine gas may be produced in water or acid as a result of the activity of fungi or bacteria on certain arsenic compounds (Wagner 1973). Dimethyl arsine has been shown to be formed by molds from arsenite with MSMA and DSMA as intermediates (Anonymous 1971; Challenger 1947).

4.3 MAJOR ENVIRONMENTAL PATHWAYS

4.3.1 Pathway 1--Atmospheric Emissions With Subsequent Transfer to Water and Soil

4.3.1.1 Sources

Emissions to the atmosphere comprise 11% of all environmental releases (see Chapter 3.0). The most significant sources are the thermal processes of fossil fuel combustion and copper smelting and nonpoint source drift from pesticide application. The forms released are primarily arsenic trioxide from the combustion processes and the various forms in pesticides (primarily MSMA and arsenic acid) depending upon which pesticides are used. These processes could be considered sources of at least localized arsenic pollution of soils and water as a result of fallout or rainout. The contribution of these sources to national ambient levels of arsenic in the atmosphere is unknown, particularly in comparison with the contribution of natural sources. This pathway focuses on transfers between the atmosphere and soil or water and only briefly considers atmospheric fate.

4.3.1.2 Atmospheric Transport

Arsenic does not appear to persist in the troposphere for a long time. A mean residence time of 9 days was estimated for atmospheric arsenic (Walsh *et al*. 1979), with losses due to transport via fallout or rainout. Arsenic may persist longer in areas with low precipitation or at times with limited atmospheric mixing.

Arsenic is released from combustion zones primarily as a vapor of arsenic (III) trioxide, As_2O_3 (see Chapter 3.0). Upon release, it adsorbs onto particulate matter and accordingly is most concentrated in particulates of small diameter. This adsorption occurs as the result of the greater surface area to volume ratio of small particulates and, according to one source (Coles *et al*. 1979), the affinity of arsenic for sulfur. Coles *et al*. (1979) found that the concentration of arsenic in fly ash fractions corresponding to mean aerodynamic diameters of 18.5 µm, 6.0 µm, 3.7 µm and 2.4 µm increased as follows: 13.7, 56, 87 and 132 µg/g. (Concentrations vary considerably by coal type, source, and combustion efficiency. Note the significantly higher levels measured and reported in Appendix C.) Coles noted that the elements associated with sulfide ores volatilize during combustion and recondense on smaller particulate matter.

The atmospheric lifetime and dispersion of arsenic are dependent upon local meteorology and size distribution of particulates. Dry deposition and wet fallout are two processes that remove arsenic from the atmosphere. On contact with alkaline media, the trioxide form is converted to arsenite, $AsO(OH)^-$, and in acids to arsenous acid, H_3AsO_3 (Mushak *et al*. 1980). Raigaini *et al*. (1977) measured the trace metal contamination of local soils and grasses near a lead smelter. The surface soils and grass contained enriched levels of arsenic and other metals; resuspension of tailings dust, in addition to direct emission of aerosols from the smelter, was identified as a major source of arsenic.

Fisher *et al*. (1979) researched the solubility and concentration distribution of arsenic on coal fly ash particulates by leaching with aqueous media, at pH 7.3. The mass of arsenic per gram of fly ash residue was approximately 120 µg over the particulate range of 0.03 µm to 5 µm. These concentrations were soluble at pH 7.3, increasing in solubility in particulates greater than 0.4 µm (solubility of about 12 µg/g) as compared with particulates smaller than 0.2 µm (solubility ~4 µg/g). This result would suggest that significant movement of arsenic into land or surface water could occur in the vicinity of an atmospheric point source from fallout of large particulates.

Crecelius *et al*. (1975) found that the major man-made contributor to arsenic concentrations in Puget Sound was a copper smelter. The annual arsenic loading to the Puget Sound was estimated as follows:

Incoming seawater	5.8×10^8 g/yr
River influx	3.0×10^7 g/yr
Precipitation	7.7×10^7 g/yr
Smelter effluent	4.0×10^7 g/yr
POTW effluent	7×10^5 g/yr
Drydock operations runoff	9×10^5 g/yr

The amount of arsenic released annually in the smelter effluent to surface water is now estimated at less than 1 kkg (see Chapter 3.0); this reduction indicates that wastewater treatment processes have become more effective over the past few years. Air emissions from the smelter were not quantified in this study; however, that rain samples in the vicinity of the smelter contained 17 µg/l of arsenic as compared with 0.4 µg/l background levels indicates a significant contribution. The percentage of arsenic input into Puget Sound due to atmospheric fallout is estimated to be 10.5%. Analysis of cores of contaminated sediment revealed 34% of the arsenic associated with iron and aluminum oxides; less than 10% was associated with easily oxidizable organic matter.

In a study of emissions from a copper smelter (with arsenic production facilities) in Sweden, Landau (1977) measured arsenic levels in various environmental media (see Table 4-3). The plant emitted an estimated 150 kkg/yr of arsenic to water, 25 kkg/yr to air and 90 kkg to soil. These air emissions are lower and the water discharges higher than the estimated releases from the U.S. ASARCO smelter (210 kkg to air; <1 kkg to water; 1,200 kkg to land: see Chapter 3.0). Although the distribution among environmental media and total release of arsenic to each differed for the two smelters, the Swedish data indicate the levels and extent of arsenic contamination associated with such sources.

High soil levels of arsenic have been reported in the vicinity of U.S. smelters. Levels of up to 150 mg/kg were measured in the soil surface layer within 1.6 km from a Montana copper smelter and decreased to background levels over a 16-km distance (Miesch and Huffman 1972). Levels of up to 380 mg/kg were detected in the soil around the ASARCO smelter in Washington and elevated levels were present as far as 40 km downwind (Crecelius et al. 1975). The ASARCO concentration was significantly higher than the levels measured in Landau's (1977) study; the difference may have been due to the smelter's larger volume (eight times that of the Swedish plant) of annual arsenic releases to the atmosphere.

The annual rate of arsenic deposition onto land and water from a coal-fired power plant (of unidentified capacity) over 56 square kilometers was estimated at 2.8 mg/m assuming a 99% electrostatic precipitator efficiency (Rancitelli et al. 1974). If one assumes that no transport out of the soil is occurring, this deposition would increase the soil concentration by 0.2 mg/kg in the top 7.6 cm of soil each year.

Another potential source of arsenic emissions to the atmosphere is from municipal incinerators. Greenberg et al. (1978) investigated the emissions from two Washington D.C. incinerators and found that >75% of the total arsenic released was adsorbed onto particles with diameters <2 um at concentrations of 170-490 µg/g (av = 310 µg/l). The arsenic released did not contribute significantly to the total annual atmospheric releases in the metropolitan area. Coal combustion was thought to be a much more significant source.

Very little information was available concerning deposition onto soil or water from emissions of cotton ginning plants. Deposition patterns are expected to be different from those from other sources of arsenic for two reasons. Arsenic is released adsorbed onto particulates composed of plant foliage, lint, and dirt with 99% >25 µm in diameter according to Herring (1973) and >3 µm according to the results of EPA tests (Suta 1980). Most of the particulates are expected to settle out within the vicinity of the gin (Suta 1980). Also emissions occur only over a 2- to 4-month period during the year as opposed to the year-round releases from smelters and coal-combustion facilities.

TABLE 4-3. TOTAL ARSENIC CONCENTRATIONS IN VARIOUS MEDIA IN VICINITY OF A COPPER SMELTER

Medium	Distance from Smelter	Concentration
moss	2-3 km	40-60 mg/kg
suspended dust	1-2 km	<1 μg/m
falling dust	not given	25 g/100 m^2 (max)
	not given	3 g/100 m^2 (average)
soil	maximum levels in vicinity	30 mg/kg
barley	vicinity	0.06 mg/kg
	40 km	0.01 mg/kg
mammals	vicinity	20-200 mg/kg
river	vicinity	500 μg/1
	30 km downstream	5 μg/1
aquatic organisms	400 km	above natural background background levels
sediment	1,000 km	above natural background background levels

Source: Landau (1977).

4.3.1.3 Summary

Atmospheric emissions of arsenic from smelting, coal combustion and cotton ginning processes may lead to localized high concentrations of arsenic in surrounding soils and surface water and contribute to high urban atmospheric levels. In the combustion processes, the higher arsenic levels are associated with particles with diameters <2 μm. Most particulates from cotton ginning processes are at least > 3 μm in diameter. Larger particulates would tend to be deposited in the immediate vicinity of sources. In Puget Sound, atmospheric fallout contributed 10.5% of the total arsenic loading and the major source of the atmospheric levels was the ASARCO smelter plant. Soil levels in the vicinity of airborne sources have been reported as high as 380 mg/kg. An atmospheric arsenic residence time of 9 days has been estimated. This persistence estimate is more applicable to small-sized particulates, which are widely dispersed, than to larger sized particulates, which would tend to fall out more rapidly in the immediate vicinity of the source.

4.3.2 Pathway 2--Solid Waste Disposal and Migration Into Ground Water

4.3.2.1 Sources

Most of all environmental releases of arsenic, 81%, are made to land in the form of solid wastes. The most significant sources are disposal of fly ash from fossil fuel combustion, solid waste resulting from smelting processes (primarily of copper) and mineral production (i.e., boron), over-burden from surface mining, landfilling of municipal solid wastes, and direct application to agricultural land and lawns as a pesticide. This last source is treated separately in Pathway 5, Purposeful Releases.

The forms of arsenic most commonly released to land are arsenate and arsenite (conversion products from arsenic trioxide on contact with aqueous media), and the various sulfur-containing arsenic complexes found in coals and ores.

4.3.2.2 Fly Ash Disposal

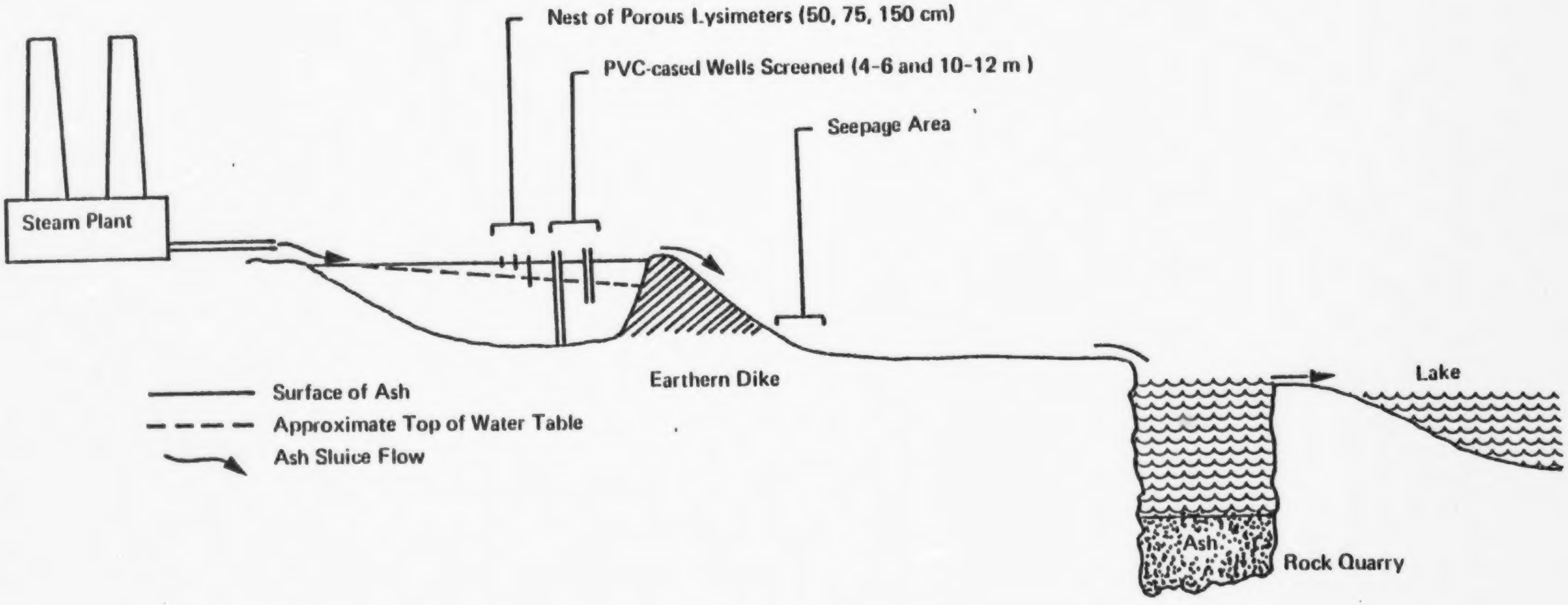
The solid waste resulting from fossil fuel combustion--fly and bottom ash--is a significant potential source of arsenic release to the environment. This is due to the high surface area associated with the waste, present disposal methods (lagoons or landfill), usually uncontrolled, and sometimes high concentration of trace elements in the waste. Research on the movement of arsenic out of disposal sites into ground water and surrounding soil is relatively recent and much of the work is in progress. Over the next few years, however, the results of disposal site monitoring studies (by the Oak Ridge National Laboratory and others) will become available.

In laboratory experiments investigating leaching from fly ash, approximately 7% of the total arsenic in a fly ash sample was released into a spring water solution (pH 8.1) after 7 days (Turner et al. 1978). Distilled water (pH 5.9) was ineffective at removing the element. Theis and Wirth (1977) found a similarly low solubility at neutral pH. Assuming the rate is representative of an average disposal site, then an estimated 980 kkg arsenic is leached from the 14,000 kkg of arsenic disposed of in fly ash annually. This number does not represent the amount of arsenic entering ground water, however, due to adsorption of mobile arsenic onto soil before it reaches the water table.

Arsenic concentrations in ash pond discharge and ground water often exceed drinking water standards due to the alkaline conditions present in ash disposal ponds (Dressen et al. 1977) which are conducive to mobility of both arsenate and arsenite (Turner et al. 1978). In addition, the high ratio of associated surface area to volume in fly ash contributes to greater potential for leaching. The arsenic form in fresh dry fly ash is predominately As_2O_3; however contact with water during sluicing produces arsenous acid, H_3AsO_3 and the arsenites, $H_2AsO_3^-$ and $HAsO_3^{-2}$. Since the environment of ash ponds and the surrounding ground water is usually low in oxygen, the trivalent form prevails once the fly ash has been deposited. Very slow oxidation to arsenate, however, may occur. Adsorption onto fly ash solids and coprecipitation with hydrous iron oxides removes some of the arsenic from solution (Turner et al. 1978).

Figure 4-8 depicts the process for fly ash disposal following generation and includes arsenic concentrations, both total and percent trivalent, in each step (Turner et al. 1978). Soil characteristics were not given. The trivalent form was most significant in the fly ash pond and relatively low at the point of release to the environment, probably because of aerobic conditions in the overflow area. In another study, Cherry and Guthrie (1977) found arsenic distribution in a fly ash basin as follows: 27 mg/kg in the sediments and 0.08 mg/l in water at pH 7.1. Turner et al. (1978) measured deep well water concentrations as high as 1.6 mg/l total arsenic, with 88% present in trivalent form. The deeper wells (12 m deep) had higher concentrations than shallower wells (4 m deep).

In a study of coal fly ash disposal at a porous sandy site in Michigan (Theis et al. 1978), arsenic was present in the fly ash at 1,200 mg/kg. Arsenic concentrations were measured in surrounding soil and ground water. Soil levels were 6.5 mg/kg near the disposal lagoon, 1.7 times greater than background levels measured 500 m away. Levels in soil at its interface with the water table were relatively uniform, 4 mg/kg to 5 mg/kg over the distance 100 m to 600 m from the lagoon. Arsenic levels in ground water were significantly lower than the concentrations within the lagoon, 50 µg/l (at 100 m away) as compared with approximately 5,000 µg/l in the disposal site. Ground water levels reflected changes in practices at the plant; a 50% reduction in operation was associated with a reduction in ground water arsenic to 30 µg/l.


Nest of Porous Lysimeters (50, 75, 150 cm)
PVC-cased Wells Screened (4-6 and 10-12 m)
Seepage Area
Steam Plant
Earthern Dike
Lake
Ash
Rock Quarry
Surface of Ash
Approximate Top of Water Table
Ash Sluice Flow

	Coal µg/g	Fly Ash µg/g	Lysimeters µg/l	Wells µg/l	Seepage µg/l	Quarry Inflow µg/l	Quarry Outflow µg/l
Total As	16–61	97–313	1.5–9.3	32–1590	50–177	30–530	120–300
%As(III)			40–100	12–88	24–58	(36)	(4)

Source: Turner *et al. (1978).*

( ) Single analysis

FIGURE 4–8 SCHEMATIC OF ASH DISPOSAL AREA ON THE OAK RIDGE RESERVATION, AND SUMMARY OF THE RANGES OF ARSENIC CONCENTRATIONS IN SELECTED SAMPLES

In a survey of a total of 11 ash disposal pond effluents, Turner *et al*. (1978) found a wide variability in pH, arsenic concentration and speciation. Table 4-4 summarizes his results. It is evident that general extrapolation from one fly ash disposal study would not give a realistic estimation of the national contribution of arsenic from fly ash.

4.3.2.3 Mining Activity

Presently, the only site in the U.S. where arsenic is produced is a copper smelter in Tacoma, Washington. However, arsenic at low levels may be released from other metals, mining and smelting sites. Abandoned gold and stibnite mines have been studied with regard to the heavy metals released to surface and ground waters. Unlike the heavy metals that tend to be solubilized by acid mine drainage, arsenic is less soluble at low pH values and is prone to adsorb onto the oxidized Fe_2O_3. Stach et al. (1978) found that arsenic as FeAsS associated with old gold deposits, was being oxidized by bacteria (*Ferrobacillus* and *Thiobacillus*) to AsO_4 and AsO_3 and then coprecipitated with iron and aluminum oxides and calcium. The ground water of the area contained arsenic levels thought to be due to water percolating through the old mine tailings and arsenic-contaminated stream sediments; a high correlation was noted between Fe and As levels.

Hawkins (1976) came to similar conclusions to explain the concentration of arsenic in ground water near an old gold mining district. Arsenic, as FeAsS, was being oxidized to H_3AsO_3 complexes and contributing to ground water contamination. Wagemann *et al*. (1978) found that a lake receiving tailing pond seepage from a gold mine contained 0.7 mg/l-1.5 mg/l of arsenic, whereas control lakes contained 0.01-0.07 mg/l. Penrose *et al*. (1975) determined the arsenic input to seawater and sediments near an abandoned stibnite mine. Arsenic entered the water by runoff from the mine and solubilization of mine tailings. Arsenic was detected at higher levels in the water during periods of rain (595 µg/l as opposed to 180 µg/l during dry spells). The contribution to seawater and sediments from the mine did not appear to be significant due to a lack of detectable surface accumulation of arsenic in sediment core analysis. This may have been due to other factors than low loadings, however, such as rapid transport out of the area or methylation by sediment bacteria.

The soil surrounding a gold mine mill tailings pond in which arsenic (sulfides and trioxide) was disposed, exhibited elevated arsenic levels up to 400 m away from the site (Comanor *et al*. 1974). Complexation of soluble arsenic forms by Al and Fe resulted in insoluble, inaccessible forms. Table 4-5 presents soil concentrations as a function of distance from the tailings piles. The highest concentration measured was 6,138 mg/kg (550 mg/l soluble) in the surface layer 30 m from one tailings pile. Although levels were generally highest in the surface layer of the soil, the data imply that vertical migration of arsenic may be an important process in some soils. The soil type studied by Comanor had pH 6.5-7.5 and <5% clay and silt.

TABLE 4-4. ARSENIC IN ASH POND EFFLUENTS FROM 11 PLANTS

Plant	pH	Total As (µg/l)	% Trivalent
1	7.1	48	6
2	5.7	1.9	17
3	9.8	130	5
4	9.6	80	7
5	9.6	1.4	11
6	9.8	28	8
7	NG	38	2
8	10.7	7.3	40
9	6.3	5.2	33
10	9.5	46	5
11[a]	7.9	119	4
Mean		35	12

NG = Not given.

[a] Turner _et_ _al_. (1978) field study described previously.

Source: Turner _et_ _al_. (1978).

TABLE 4-5. CONCENTRATIONS OF TOTAL AND SOLUBLE ARSENIC MEASURED IN SOIL SURROUNDING A MINE TAILINGS DISPOSAL SITE

Arsenic Species/ Depth	Concentration (mg/kg) at Distance from Site (m)				
	30	165	290	315	450
Total As: ↓					
Surface	6138	2409	670	780	169
0-15 cm	644	190	191	73	52
15-30 cm	368	93	34	16	7
30-45 cm	247	32	56	35	38
45-60 cm	798	52	24	7	39
Soluble As: ↓					
Surface	550	76	17	59	8
0-15 cm	115	32	7	Nil	Nil
15-30 cm	73	3	Nil	Nil	Nil
30-45 cm	43	Nil	Nil	Nil	Nil

Source: Comanor *et al*. (1974).

Leaching coefficients for arsenic from copper and lead slag were estimated at 0.05% and 0.1%, respectively, of the total amount of arsenic initially present in the slag (Twidwell 1980). These coefficients were based on observations with distilled water leachate (pH 7) and are presumably for total arsenic, with no differentiation between forms. No other detail was provided. It is worth noting that Theis and Wirth (1977) found minimal solubility of arsenic fly ash at neutral pH.

Approximately 16,000 kkg of arsenic were estimated to be deposited onto land each year in slag from arsenic, copper, lead, iron and steel, zinc and aluminum production processes (see Chapter 3.0). On the basis of Twidwell's estimates, approximately 4 kkg of arsenic leaches from copper wastes annually and 8 kkg from all other metals wastes. The latter estimate assumed the higher coefficient derived for lead was applicable to other metals for which no measurements were available. There is a great deal of uncertainty associated with these estimates due to the potential for much higher leaching rates under certain environmental conditions (see 4.2, soil).

Even though arsenic in smelting wastes is initially in trivalent form (following conversion from arsenic trioxide), the most common water soluble form found in soil at waste disposal sites was arsenate. at a ratio of 8 or 9 to 1 (Porter and Peterson 1977). In some soil samples, dimethylarsine made up to 10% of the total arsenic levels (These data are presented in greater detail in Section 4.4.7). The presence of methylated As forms was thought to be due to microbial activity either methylation of free arsenite or reduction of arsenate. The authors expected microbial activity at mine tailings disposal sites to be minimal due to low levels of organic matter and essential nutrients, thus, the presence of methylarsine was not anticipated.

4.3.2.4 Landfills

In a national study of subsurface migration of pollutants at selected industrial waste land disposal sites (Geraghty and Miller 1977), arsenic was found to be present at 37 out of 50 sites and at 30 sites to have migrated to ground water. The sites were either active for more than 3 years or abandoned, including lagoons and landfills from eleven East Coast states. Concentrations in ground water at distances of 30.5 m to 152.5 m from the disposal site ranged from 0.04 mg/l to 5.8 mg/l (four samples) (see Table 4-6). All other measured concentrations were lower. Sampling was not conducted any further laterally than 610 m from any site to indicate the extent of arsenic migration. Arsenic was the second most frequently encountered inorganic (after selenium) to exceed the drinking water standard of 0,05 mg/l.

Eichenburger et al. (1978) studied inorganic constituents of hazardous solid waste discharges of five Class I landfill sites in Los Angeles. Seventeen industrial waste streams were studied. Arsenic was found to

TABLE 4-6. ARSENIC CONCENTRATIONS IN GROUND WATER NEAR INDUSTRIAL WASTE DISPOSAL SITES

Site	Concentration in in Groundwater (μg/l)	Distance From Disposal Area (m)	Total Depth (m)
Connecticut	40	30.5	5.2
Indiana	5,800	36.6	NA
Massachusetts	650	6.1	4.0
New Hampshire	120	61.0	12.2
New Hampshire	30	152.5	11.9

Source: Geraghty and Miller (1977).

be the third lowest of the 17 metal species sampled in terms of estimated total daily deposition (8×10^3 g/day) and most of the arsenic in the waste was produced by chemical industries. Less than 5% of the metal was detected in dissolved form. The average total concentration was 0.4 mg/ml in sludge.

Arsenic (as soluble arsenate) was detected in ground water samples in the vicinity of two municipal landfills in Babylon and Islip, NY (Kimmel and Braids 1980). In the immediate vicinity of the sites, ground water concentrations measured 14 µg/l and 29 µg/l, respectively, at the two sites. Further from the site, concentrations in the leachate plume were lower at 1 µg/l (0.75 km away) and 2 µg/l (1.5 km away). The source of arsenic was thought to be from contaminated phosphorus and selenium salts and, less so, from disposal or use of arsenical pesticides. Arsenic levels in presumably uncontaminated, "native" ground water samples ranged from not detectable to 11 µg/l. The fact that the physical reach of high arsenic levels at this location was not significant was probably due to low concentrations in the solid waste disposed of at the landfill.

Arsenic disposed of at land disposal sites may pose a long-term hazard, infiltrating ground water through subsurface migration. Arsenical grasshopper bait (~50 lb arsenic trioxide) buried in soil between 1934 and 1936 in Perham, MI, caused poisoning of 11 people approximately 40 years later (U.S. EPA 1975). Exposure occurred following drilling and use of a drinking water well at a distance of approximately 9.2 m from the disposal site. The disposal site was 2.1 m deep and the well, 9.4 m deep. Arsenic concentrations of 21 mg/l were measured in well water samples.

An incident of ground water contamination by arsenic seepage from a World War I ammunition dump was reported in Germany (Koppe and Grebler 1968). Arsenic migrated vertically over a 5 m distance and resulted in levels of 1,000 µg/l to 10,000 µg/l in ground water over an area of 5,000 m^2. No detail was available regarding soil characteristics or by what mechanism the arsenic was transported after so many years.

4.3.2.5 Summary

Releases to land account for the largest fraction, 81%, of all environmental releases of arsenic. Sources include fly ash disposal, mine and smelter tailings ponds and landfills. There is a potential for migration of arsenic from the disposal site into surrounding soil and ground water for all sources. Leaching from fly ash is greatest under alkaline conditions, which are characteristic of disposal sites. Both arsenate and arsenite are present in the waste in a distribution dependent upon environmental conditions. There is a considerable amount of variability among sites in the arsenic concentrations associated with pond effluents, soil, and ground water and in the fraction of total arsenic present as the trivalent. The highest effluent level measured was 130 µg/l (5% trivalent); the highest ground water level was 1,600 µg/l (88% trivalent). Mining disposal sites usually have low associated pH levels at which arsenic is less mobile. However, high arsenic levels have been measured in the vicinity of gold, stibnite, and other mining sites. Concentrations as as high as 1.5 mg/l were measured in runoff; at one site, surface soil levels (total As) were 6,138 mg/kg and, at 45-60 cm deep, as high as 798 mg/kg. Arsenate, arsenite, and dimethylarsine have been detected at tailings disposal sites, with arsenate predominating (at least under aerobic conditions). Arsenic is commonly detected in ground water in the vicinity of industrial landfill sites; the highest reported level was 5,800 μg/l. There is a long-term potential for subsurface migration of arsenic into ground water as illustrated by two incidents of contamination more than 30 years following disposal of arsenic-contaminated waste.

4.3.3 Pathway 3--Direct Discharge to Surface Water

4.3.3.1 Sources

Industrial discharge of arsenic in effluents contribute only a small fraction, 3%, of all environmental releases. [Note that the 5% calculated in the Materials Balance Chapter 3.0 also includes contributions from soil runoff following pesticide application to land; this nonpoint source is not considered in this pathway]. The most significant known sources are zinc production facilities and use of arsenic-contaminated detergents. Other potential sources include natural releases from weathering of bedrock (an estimated 6,700 kkg annually) and discharges from POTWs (an estimated 1,800 kkg annually). The forms of arsenic released are unknown; however, various compounds would be expected to be present including inorganic complexes associated with phosphorus and smelter ores, simple arsenates and arsenites.

Relatively little fate information was available concerning aqueous discharges of arsenic resulting from human activities as compared with its other environmental pathways. Due to the small contribution of these releases to the total environmental loading of arsenic, it was thought appropriate to limit this discussion to available data and not supplement it with use of calculational models and other estimation techniques. Most of the available data concerned the various arsenic forms detected and their fraction of the total concentration measured in different types of water bodies. In addition, some information was available on release of sediment bound arsenic to the water column. Arsenic concentrations measured in aquatic organisms and the mechanism of uptake are also discussed in this pathway. Wastewater treatment of industrial effluents is discussed in Pathway 4.

4.3.3.2 Field Studies of Arsenic in Aquatic Systems

In a study of the ASARCO smelter's aquatic discharges (Crecelius *et al*. 1975), sediment in Puget Sound was found to contain arsenic at levels up to 10,000 mg/kg and surface seawater in the immediate vicinity of the plant to contain 1.2 mg/l. Within 1.6 km, the water concentrations were reduced to 4 μg/l, which was attributed to sorption onto sediment. Sediment levels at this location were not given. STORET data were examined for this area and the results are discussed in Chapter 6.2.

An estimated 55% of arsenic discharged to Lake Washington by a smelter was removed by sediment, primarily by sorption on or coprecipitation with iron and manganese compounds (Crecelius 1975). Some of the remaining sediment-associated arsenic was thought to have entered the lake already bound to smelting-waste particulates. In aquatic systems receiving a larger fraction of dissolved arsenic, Crecelius expected a lower amount of arsenic to end up in the sediment.

Waslenchuck (1978) analyzed arsenic speciation in the Continental Shelf waters of the Southeastern U.S. Total arsenic concentrations, averaging 1.1 μg/l, in the Georgia Bight were found to be controlled primarily by Gulf Stream intrusions. Upstream river concentrations (average 0.3 μg/l dissolved As) were found to be controlled by rainwater dilution and complexation with dissolved organic matter which prevents adsorption of arsenic on solid phase organic and inorganic substances. Arsenate was the predominant form present; arsenite and dimethyl-arsenic concentrations were an order of magnitude lower than arsenate levels and comprised approximately 20% of the total arsenic levels. Arsenate concentrations in the water column were lower than saturation levels due to processes removing the free arsenate ion from solution. The residence times estimated for total arsenic was 0.23 years in the rivers and 0.20 years in the Bight.

Following precipitation and/or adsorption of arsenic into the sediment layer of aquatic systems, there is a potential for release to the water column. Arsenic was released to water to a greater extent and at slightly faster rates from anaerobic muds than from aerobic muds (Faust and Clement 1980). The study used polluted muds from New Jersey surface waters used for recreational purposes and as a secondary water supply. By 28 days under anaerobic conditions, up to 60% of the initial 19 μg/g arsenic in mud was released to the water phase to make up water concentrations of 1.2 mg/l. At higher mud concentrations, 40% of an initial 381 μg/g in mud was released resulting in a water concentration of 12.6 mg/l. Release was greater by 70% at 25-30°C than at 20°C; but at 37°C, release was 40% lower than at 25-30°C. The greater release of iron from anaerobic as compared with aerobic muds suggested that some of the arsenic releases may be attributable to dissolution of ferrous arsenite compounds. The anaerobic muds were generally more acidic than the neutral aerobic muds, which may have been a cofacter in the difference in As release rates from the muds.

Braman and Foreback (1973) analyzed samples from surface, ground and estuarine waters in the area of Tampa, FL. Samples were analyzed specifically for arsenate, arsenite, and the methylarsonic acids. Table 4-7 reports the concentrations found. The predominant form was arsenate (V) with several exceptions. In the two freshwater lakes, arsenite (III) levels were highest and in the saline McKay Bay,

TABLE 4-7. ARSENIC COMPOUNDS IN TAMPA, FLA WATERS

Body of Water	Concentration (μg/l) As(III)	As(V)	Methylarsenic Acid	Dimethylarsenic Acid	Total
Hillsborough River	<0.02	0.25	<0.02	<0.02	0.25
Withlacoochee River	<0.02	0.16	0.06	0.30	0.42
Well water near Withlacoochee River	<0.02	0.27	0.11	0.20	0.68
Remote pond, Withlacoochee Forest	<0.02	0.32	0.12	0.62	1.06
University Research Pond, University of Southern Florida	0.79	0.96	0.05	0.15	1.95
Lake Echols, Tampa	2.74	0.41	0.11	0.32	3.58
Lake Magdalene, Tampa	0.89	0.49	0.22	0.15	1.75
Saline Waters					
Bay, Causeway	0.12	1.45	< 0.02	0.20	1.77
Tidal Flat	0.62	1.29	0.08	0.29	2.28
McKay Bay	0.06	0.35	0.07	1.00	1.48

Source: Braman and Foreback (1973).

dimethylarsenic acid was most prominent. The one ground water sample contained arsenite below the detection limits.

A similar characterization study of two Florida water bodies was conducted by Braman et al. (1977). In a golf course pond, 46% of the total arsenic content was arsenite and/or arsenate and the remaining fraction methylarsenic acid. In Lake Carroll, the total arsenic levels were comprised of arsenite and/or arsenate (75%), methylarsenic acid (6%), dimethylarsenic acid (9%), and trimethylarsine compounds (7%). The arsine compounds were not attributed to any particular source.

4.3.3.3 Bioaccumulation in Aquatic Organisms

Introduction

Bioaccumulation and biomagnification of arsenic have been addressed in several studies of both laboratory and field situations. Many of the field studies have been conducted to determine natural background levels of arsenic in organisms; since for the most part, water and sediment concentrations of arsenic were not known, bioconcentration factors can not be calculated. Some general observations can be made, however, concerning the extent to which arsenic from a variety of arsenical compounds appears to bioaccumulate in the natural environment.

- Arsenic apparently bioaccumulates to a greater extent in marine than in freshwater organisms (Penrose et al. 1975), and more in invertebrates than finfish in both environments.

- In "uncontaminated" locations and areas not proximal to any known natural source, arsenic has been found in fish and invertebrates at levels of <0.4-87.8 μg/kg (LeBlanc and Jackson 1973).

- Arsenic is not accumulated in the more toxic arsenious oxide form as readily as in arsenate form (LeBlanc and Jackson 1973); once in the tissues, the trivalent form is apparently weakly held and available for desorption (Isensee et al. 1973).

- Overall, arsenic is neither accumulated significantly in an individual organism nor biomagnified in the food chain to any great extent. Arsenic is generally found in greater concentrations in lower food chain organisms (algae, invertebrates) than in higher forms (fish, higher invertebrates); this may be due to factors such as greater surface area for absorption in the simpler forms.

Metabolism

Accumulated arsenic in higher aquatic organisms is reported to be converted to detoxified organic form and excreted. Ingested inorganic arsenic was converted by fish to organic while injected and gill-absorbed arsenic was not (Lunde 1972, Penrose 1975). Intestinal microflora were implicated as responsible for at least the first stage in bioconversion (Penrose _et al_. 1977). The extent of arsenic metabolism and excretion in fish in general and the nature of the differences between freshwater and marine species are not well understood.

Bioconcentration Factors

Four arsenical herbicides--MSMA, DSMA, arsenic trioxide and pentoxide--were tested for their potential to bioaccumulate in fish (rainbow trout, bluegill), scud, daphnia, and snails (Spehar _et al_. 1980). There was little difference between the herbicides for all aquatic organisms tested; bioconcentrations factors ranged from <1 to 20. In another study, a BCF of 4 in bluegill for arsenic trioxide was measured; a clearance half-life of less than 1 day was also estimated. Based upon these studies and additional observations made by Isensee _et al_. (1973), a BCF for arsenic in fish in general was estimated to be usually less than 10.

Reports of tissue levels of 1 mg/kg (wet weight) up to 100 mg/kg in fresh and marine species (see following section) suggest higher BCF's than those measured under laboratory conditions. This could be attributed to an equilibrium in tissue levels not being reached before termination of the experiment or more than one pathway for arsenic uptake by fish in the environment. The form of arsenic involved is assumed to be primarily inorganic (tri- and pentavalent).

Detectable accumulation of arsenic by aquatic organisms is not always evident in arsenic-polluted areas. Fish collected from a lake 21 days after application of sodium arsenite showed no significant accumulation of arsenic.

Monitoring Data

Numerous monitoring programs for arsenic levels in fish and invertebrate tissues have been conducted both on a national scale and at specific sites. The STORET data base contains 475 observations in 13 major river basins from the past 3 years (see Table 4-8). Mean levels in fish (various species) ranged from 0.1 mg/kg to 4 mg/kg with a maximum level of 700 mg/kg observed in the Western Gulf.

The National Pesticides Monitoring Program sampled freshwater fish from monitoring stations in freshwater throughout the country during 1971-1973. Maximum arsenic residues were 3.40 mg/kg in 1971, 1.70 mg/kg in 1972, and 1.24 mg/kg in 1973. Arsenic residues greater than 0.5 mg/kg were found in fish (both predatory and non-predatory) from five stations,

TABLE 4-8. AMBIENT ARSENIC CONCENTRATIONS IN FISH TISSUE -- UNREMARKED DATA IN STORET, 1977-1979

Major River Basin	Observations	Concentration (mg/kg - wet weight) Maximum	Mean
North Atlantic	3	0.2	0.2
Southeast	2	0.6	0.5
Tennessee River	10	4.0	3.6
Ohio River	18	4.0	4.0
Upper Mississippi	127	2.0	1.8
Lake Michigan	54	2.0	2.0
Missouri River	56	2.0	0.2
Lower Mississippi	99	0.8	0.1
Western Gulf	35	700.0	0.6
Pacific Northwest	56	0.8	0.3
California	4	0.2	0.1
Great	4	0.8	0.6
Lake Superior	7	2.0	2.0
Gross Analysis	475	700.0	2.6

Source: STORET Water Quality Control Information System as of November 13, 1980.

in the Tombigbee River, LA, Lake Michigan, Lake Superior, and Red River, OK; the fish from the Great Lakes had high residues more frequently than the others (Walsh et al. 1972). No information on water or sediment concentrations was available. However, STORET data for the last 5 years indicated higher than national mean levels in the Great Lakes major river basin.

The National Marine Fisheries Service surveyed the trace element content of the U.S. fishery resource (Hall et al. 1978). Two hundred four species were sampled from 198 sites in seven U.S. coastal areas. Average levels of arsenic varied widely for finfish muscles and livers, whole fish, and molluscs, with most species containing between 2.0 mg/kg and 5.0 mg/kg in all categories. Crustacea had somewhat higher levels, with most species containing between 4.0 mg/kg and 5.0 mg/kg. Water concentrations of arsenic were not given, but arsenic concentrations in seawater are believed to be rather constant, ranging between 1.0 μg/l and 1.5 μg/l (Saunders 1978, Waslenchuk 1977, 1978, Andreae 1978); these levels would indicate BCF's of up to 500.

A survey of natural background levels of arsenic in marine fish and invertebrates near a proposed mine site on the Canadian coast indicated that levels in crustaceans were higher than those in fish from the same locations (LeBlanc and Jackson 1973). The range of levels for all organisms tested was 0.4 to 27.8 mg/kg. The median levels in the crab, Cancer magister (6-8 mg/kg), were about two times greater than the levels in fish. The authors postulated that the arsenic in these organisms, particularly the levels in crustaceans, could not be present as an arsenious oxide or any other known toxic form because of the high levels present.

One reason for higher levels in invertebrates in a number of studies may be that benthos, and other detritus, suspension and filter-feeders have access to several sources of arsenic in the water column, particulate matter and in sediment in which concentrations often exceed water levels.

Wagemann et al. (1978) measured concentrations of arsenic in aquatic invertebrates, macrophytes, sediments, and water in the vicinity of Yellowknife, Canada. In arsenic-contaminated lakes (a consequence of gold mining activities), arsenic concentrations ranged from 700 μg/l to 5,500 μg/l in water, 6 mg/kg to 3,500 mg/kg in bottom sediments, 150 mg/kg to 3,700 mg/kg in macrophytes, 700 mg/kg to 2,400 mg/kg in zooplankton, and <1 to 1,300 mg/kg in other invertebrates.

Arsenic concentrations were measured in clams, plankton, and sediments from Lake Washington and Sardis Reservoir located in rural, agricultural areas in Mississippi (Price and Knight 1978). Samples were gathered monthly from October 1975 to May 1975. Mean concentrations in mg/kg wet weight were 2.99 in sediment and 21.74 in plankton. The ranges and means of arsenic in eight species of clam were also reported. Means ranged from 0 mg/kg to 0.36 mg/kg (wet weight) and the total range was <0.01 mg/kg to 1.39 mg/kg.

4.3.3.4 Summary

Only a small fraction (3%) of the total industrial releases of arsenic are made directly to surface water. Other sources include natural weathering of arsenic-containing minerals and soil and discharges from POTWs. Sediment serves as a reservoir for a significant fraction of arsenic discharged into both freshwater and estuarine systems. Other controlling factors on concentration are rainwater dilution and dissolved organic matter content. Sediment-bound arsenic may be subsequently released to the water column following methylation by both aerobic and anaerobic sediment microbial populations..

Arsenate was the predominant form in a number of freshwater systems; arsenite was the predominate form in two lakes and dimethylarsenic acid in an estuary. Other arsenic compounds detected in freshwater bodies were methylarsonic acid and trimethylarsine. Bioconcentration of arsenic has been detected in marine and freshwater fish and invertebrate species. Fish tissues generally contain less than 10 mg/kg while invertebrates (i.e., shellfish) sometimes accumulate higher levels. Arsenic does not appear to biomagnify in the food chain.

4.3.4 Pathway 4--Wastewater Treatment

4.3.4.1 Sources

Arsenic is present in the process waters of numerous industries and in the influent to Publicly Owned Treatment Works (POTWs). Table 4-9 list industries for which the Effluent Guidelines Division has found levels of arsenic in the raw waste; the associated concentrations are also presented. Some of the highest mean levels of arsenic (total) were found in the raw waste of inorganic pesticide manufacturers, nonferrous metals manufacturing operations, and steam electric power plants. According to Chapter 3.0 (Materials Balance), approximately 1,800 kkg of arsenic enters U.S. POTWs annually. Up to 1,050 kkg of that amount may come from urban runoff; less than 60 kkg appears to be discharged directly to POTWs by industries.

The degree to which arsenic is removed from municipal and industrial wastewaters and thus the ultimate distribution of arsenic between POTW aqueous effluents and sludge depends upon the type of treatment involved. Primary and secondary treatment techniques are not very effective at removing arsenic. The efficiency of activated sludge treatment (secondary process) is variable. Tertiary treatment techniques are generally quite effective. The following section discusses (1) primary and secondary treatment of arsenic particularly in POTWs and (2) tertiary treatment techniques in general.

4.3.4.2 Primary and Secondary Treatment

According to the Materials Balance (Chapter 3.0), POTWs are virtually ineffective at removing arsenic from water. This conclusion is based on

TABLE 4-9. OCCURRENCE OF ARSENIC IN RAW INDUSTRIAL WASTEWATER[a,b]

	Raw Wastewater					
	Concentration (μg/l)			Loading [c] (kg/d)		
Industry	Minimum	Maximum	Mean	Minimum	Maximum	Mean
Coal mining	< 2	250	< 86	0	2.3	0.32
Textile mills	NA	200	10[d]	4.2×10^{-5}	0.29	0.018
Timber products processing	BDL	14,000	10[e]	0	0.46	0.00086
Petroleum refining	3	480	< 20[d]	0	1.9	0.12
Paint and ink formulation	BDL	800	73	0	0.0033	0.00012
Gum and wood chemicals	< 10	110	< 50	0.00045	0.38	0.82
Auto and other laundries	ND	1,600	68	6.1×10^{-5}	0.095	0.014
Porcelain enameling	5	2,800	960	0.00061	0.39	0.077
Pharmaceutical manufacturing	ND	120	13	0	0.01	0.088
Ore mining and dressing	< 1	110	< 20	0	NA	< 0.74
Steam electric power generating (condenser cooling system)	4	35	7	0.0004	0.008	0.0017
Steam electric power generating (water treatment)	NA	NA	9,500[e]	6×10^{-5}	570	1.4
Steam electric power generating (boiler or steam generator blowdown)	NA	NA	NA	NA		NA
Steam electric power generating (maintenance cleaning)	5	310,000	41	4.1×10^{-8}	0.78	0.012
Steam electric power generating (ash handling)	BDL	74	9	0.00017	0.88	0.20
Steam electric power generating (air pollution control devices)	< 4	300	150	0.00014	8.6	0.54
Steam electric power generating (drainage)	NA	NA	NA	NA	NA	NA
Inorganic chemicals manufacturing	NA	956[f]	NA	0	60	12
Coil Coating	75[g]	75[g]	75[g]	0.004	0.135	0.036
Foundries	ND	160	29	0	0.19	0.08
Nonferrous metals manufacturing	ND	310,000	13,000	0	NA	689
Iron and steel manufacturing	NA	440	120	0	NA	22

[a] Information contained in this table was obtained from Volume II of the Treatability Manual - U.S. EPA (1980b).
[b] NA - not available; ND - not detected; BDL - below detection limit.
[c] Pollutant loadings determined by multiplying mean pollutant concentration by industry wastewater discharges. Where mean is not available 1/2 the reported maximum was utilized.
[d] Median, not average.
[e] Average of medians reported for various industry segments.
[f] Average of maximums reported for various industry segments.
[g] One sample.

the results of a 20-plant study (see Table A-7 in Appendix A). Due to the high detection limits (~50 μg/l) associated with the analytical methods used at some of the plants which often exceeded arsenic levels in the influent, it was not possible to detect a decrease in arsenic following treatment. This analytical problem, combined with the observation that arsenic did accumulate in the sludge of POTWs, indicates that at least some removal is occurring.

In a separate study of U.S. POTW effectiveness, arsenic was reported to be removed at efficiencies of 12-43% in primary treatment and 43-85% in secondary treatment (Gilbert 1980). In another study, arsenic concentrations in domestic sewage sludge from ten U.S. POTWs were 5 mg/kg to 46 mg/kg (assumed dry weight) (Jones and Lee 1977) as compared with the average 0.2 mg/kg (dry weight) reported in Chapter 3.0. Therefore, it appears that POTWs can be somewhat effective at removing arsenic; however, the efficiency of removal varies considerably among plants. This subject requires further investigation.

In a survey of pollutant concentrations in the final effluents of municipal waste dischargers in California, arsenic was detected at five plants. Average effluent concentrations ranged from 1 μg/l to 280 μg/l, with four out of five averages less than 15 μg/l (CWRP 1977). There was no obvious relationship between high levels and the level of treatment or plant flow rates.

4.3.4.3 Tertiary Treatment

Removal of arsenic by tertiary treatment is best achieved by adjusting the pH of the commonly acidic wastewater to alkaline conditions; in wastewaters containing other heavy metals, removal of arsenic is partially achieved by coprecipitation with these metals. The most common and effective treatment technique is to remove arsenic by coagulation (with $FeCl_3$ or Fe_2So_4), sedimentation, and filtration. Up to 95% of the arsenic initially in the waste stream may be removed by this treatment technique. The fraction removed is then disposed of in solid or semi-solid form to land.

Week (undated) studied the removal of metals by tertiary wastewater treatment processes such as (1) "high lime" treatment, in which lime is added to bring the pH to 11, $FeCl_3$ is added as a coagulant, and the effluent is fed through sand and carbon columns; and (2) "alum" treatment, in which aluminum sulfate and lime are introduced to promote coagulation followed by carbon and sand filtration. For arsenic, the alum treatment was most effective although removal of arsenic in the carbon columns was three times more effective following lime treatment than alum treatment. Table 4-10 summarizes treatment efficiencies for arsenic associated with different treatment methods.

In a water treatment process plant on the Kansas River, Angino et al. (1970) reported lime softening to be effective at removing 85% of the water's total arsenic concentration. However, other sources contradict

TABLE 4-10. ARSENIC REMOVAL EFFICIENCY OF WASTE TREATMENT METHODS

Treatment	Concentration (mg/l) Initial	Concentration (mg/l) Final	% Removal
Lime Softening	0.2	0.03	85
Charcoal Filtration	0.2	0.06	70
Ferric Sulfide Filter Bed	0.8	0.05	94
Coagulation with Ferric Sulfate	25.0	5 or less	80 or more
Coagulation with Ferric Chloride	3.0	0.05	98
Precipitation with Ferric Hydroxide by Ferrous Sulfate & Lime Coagulation with Settling only	362 (as Arsenic Oxide)	15-20 (as Arsenic Oxide)	94-96
Ferrous Sulfide Filter Bed, Bone Carbon & Settling	0.8	0.05	94
Ferrous Sulfide Filter Bed, followed by Sand & Coke Filtration	0.5	trace	
Chemical Coagulation, Sedimentation, and Filtration	1.0-2.0	0.05-0.5	80-95

Source: Week (undated).

this finding. The Federal Water Pollution Control Administration (1968) reported that typical water treatment plant processes--coagulation, sedimentation, rapid sand filtration--were ineffective at removing arsenic. Arsenic concentrations measured in finished drinking water are generally very low across the entire U.S. (see Section 5.2), an observation indicating either low initial concentrations or effective treatment techniques.

4.3.4.4 Summary

Arsenic is removed from wastewater during primary, secondary, and tertiary treatment. Tertiary treatment, especially coagulation/precipitation and lime softening processes, are very effective, usually >85%, at removal. However tertiary treatment is relatively uncommon compared to primary and secondary treatment, and is usually limited to industrial applications. The effectiveness of primary and secondary treatment in removing arsenic is unclear and appears to be quite variable within different systems. POTWs, the majority with only primary and secondary treatment processes, vary in efficiency from 0% up to 85%. The overall effectiveness of U.S. POTWs on a national scale requires further investigation.

4.3.5 Pathway 5--Pesticidal Uses

4.3.5.1 Introduction

Approximately 10,250 kkg of arsenic are used each year in the manufacture of pesticides, primarily MSMA (29% of total production), arsenic acid (28%), DSMA (6%), cacodylic acid (7%) and others including sodium arsenite (making up remaining 30%). Almost all of the amount produced is expected to be released to the environment during use. An estimated 15% of all releases is expected to be emitted to air, 81% to land, and the remainder to surface water (including runoff from soil surfaces). Most arsenic forms are expected to be equally distributed in the environment; however the use of sodium arsenite as an aquatic herbicide may make surface water a significant receiving medium for this form.

Arsenic pesticides can be classified into inorganic and organic arsenicals. Of the first group, arsenic acid (H_3AsO_4) is used to defoliate and desiccate cotton; sodium arsenite, as already mentioned, is used as a lake herbicide; lead and calcium arsenates are insecticides used against leaf chewing insects; and chromated or ammoniacal copper arsenate are common wood preservatives. The organic arsenicals include methanearsonic acid (MAA) and its mono- and di-sodium salts (MSMA and DSMA, respectively), and cacodylic acid (CA or dimethylarsinic acid) (Woolson 1977). Organic arsenicals are mostly used against postemergent weeds and applied as a foliar spray. Arsenic is also present as an impurity of both natural and synthetic phosphate fertilizers (see Section 4.3.5.3).

4.3.5.2 Field Studies on Arsenical Pesticides

The total amounts of inorganic and organic arsenical pesticides produced annually are approximately equivalent; however, organic forms are usually applied at lower concentrations than are inorganic forms (Sandberg and Allen 1975). MSMA and DSMA are often applied at rates of less than 10 kkg per hectare in one application while sodium arsenate and, in the past, lead arsenate, are applied in excess of 100 kg per hectare. Most of the agricultural sites, such as orchards, with high residue levels of arsenic were treated with inorganic arsenicals.

Pesticide Drift

Most arsenicals are applied in a spray form. Although the chemical forms used are not volatile, there is a potential for drift during treatment to non-target areas, both soil and water. MSMA levels in the water in a sprayed irrigation ditch reached up to 860 μg/l immediately following treatment but declined to less than 50 μg/l within 2 hours (Salman et al. 1972). Loss of arsenic from surface water is dependent upon water volume, flow rate and other factors described in Pathway 3. The total amount of arsenic pesticide lost through drift during normal application procedures is expected to be minimal (Sanberg and Allen 1975).

Adsorption

On reaching the soil, the chemistry of organic and inorganic arsenicals, regardless of the form, is essentially the chemistry of arsenate, provided that aerobic conditions prevail (Walsh and Keeney 1974). Activity of arsenicals is reduced due to immediate adsorption by soil colloids (Hiltbold 1975) and formation of insoluble salts (Walsh and Keeney 1974). The water-soluble fraction of cacodylic acid decreased over a period of 8 weeks and was replaced by a less soluble form of arsenic associated with aluminum (Hiltbold 1975).

Adsorption of arsenic is highly correlated with soil clay for all arsenic content ($r = 0.81-0.96$; Hiltbold 1975). In clay soils with a high anion exchange capacity (e.g., high kaolinite content), arsenic (as arsenate) can be adsorbed at quantities up to 1% by weight of the clay (Pax 1973) depending on the presence of other competing anions (especially $SO_4^=$, NO_3^-). Addition of these competing substances in fertilizer could also displace arsenic already adsorbed in the soil. Clay soil adsorbed up to 2,270 μg As per gm of soil at equilibrium (Wauchope 1974). Soils high in iron or calcium and also organic soils are likely to fix arsenic in insoluble form through complexation and chelation (Cooper et al. 1932). Examples of soils with a high arsenic fixing capacity include red and yellow podzols, latosols, arid soils, and limestone soils. Subsoils tend to have a greater holding capacity for arsenic than do surface soils, presumably due to higher concentrations of clay and iron oxides (Hiltbold et al. 1974). In all soils there is always potential for subsequent resolubilization of the fixed arsenic following changes in soil conditions including a rise in pH in high iron soil, a drop in pH in lime soil, or a change in redox potential.

Leaching

A number of studies have been conducted on the leaching of arsenical pesticides through agricultural soils. Table 4-11 summarizes the results from field studies on inorganic and organic arsenicals. In most soils, some leaching occurred within the top 30 cm of soil, more so in sandy soils than in clay or loam soils. No detectable arsenic was found deeper than 90 cm. Environmental factors instrumental in the mobility of arsenic include the initial arsenic concentration, rate of precipitation and soil characteristics such as adsorptive capacity. Soil pH does not appear to have a significant influence on dissociation and potential mobility of organic arsenicals (Hiltbold 1974). However, pH may have some influence on the availability of iron and aluminum in soil which in turn influence arsenic mobility.

Surface Runoff

Transfer of soil surface-adsorbed arsenic to surface water via erosion and surface runoff may occur during precipitation. The amount of arsenic transferred in surface runoff depends on the local terrain (i.e., slope), vegetative cover, and rainfall patterns. An annual erosion rate of 2.78-19.72 tons of soil per acre was estimated for agricultural soils in Missouri (rainfall = 40 inches) (Brady 1974). For soil with an arsenic level of 10 mg/kg, an estimated 60-425 g of arsenic per hectare would be lost annually through surface runoff. An estimated 7% of the total amount of arsenical pesticides applied to land is transferred to surface water each year (Chapter 3.0).

Oxidation of Organic Arsenicals

The organic arsenicals may undergo oxidation due to microbial activity in aerobic soils. A rate of oxidation for MSMA was estimated at 2% per month in light textured, low organic soils and 10% per month in organic clay soils (Hiltbold 1974). Forty-one percent of initially applied cacodylic acid was oxidized to arsenate and CO_2 in a 24-week period (Woolson and Kearney 1973).

Volatilization

Some of the arsenic applied to agricultural soils in both organic and inorganic form may be lost through volatilization of methylated forms resulting from microbial transformations. In some cases the amount of arsenic lost by volatilization is significant. Approximately 35% of cacodylic acid was lost in a 24-week period in aerobic soil and 61% in anaerobic soils, possibly as dimethyl arsine (Woolson and Kearney 1973). The rate of transformation is dependent on the environmental factors influencing microbial activity: organic content, pH, moisture content, microbial species present. Even though the methylated forms of arsenic are highly volatile, the transfer from soil to the atmosphere may be slow in soil with a nonporous texture or with heavy vegetative growth. In these situations, oxidation to the more immobile arsenate form may occur before the arsenic moves very far.

TABLE 4-11. FIELD STUDIES ON LEACHING OF ARSENICAL PESTICIDES THROUGH SOIL

Form Applied	Application Rate	Soil Type	Result	References
Arsenic	NG[a]	clay adobe	Concentrations of 441 mg/kg[b] in surface 20 cm of soil but 10 mg/kg in next 20 cm.	Jones and Hatch (1937)
Arsenic	NG	silt loam	Concentrations of 61 mg/kg in surface 20 cm of soil but 8 mg/kg in next 20 cm.	Jones and Hatch (1937)
Pb arsenate	NG	orchard soil	Arsenic not leached below root zone	Benson (1968)
Pb arsenate	NG	orchard soils	Water soluble arsenic concentrated in top 15 cm soil; little found below 61 cm.	Vandecaveye *et al.* (1936)
Arsenic trioxide	1,120-8960 kg/ha	silt loam	No arsenic residues below 36 cm deep.	Arnott and Leaf (1967)
Sodium arsenite	90-180 kg/ha	sandy soil	Arsenic leached to 38 cm deep 3 years after application.	Steevens *et al.* (1972)
Sodium arsenite	760 kg/ha	sandy soil	Arsenic leached to 68 cm deep 3 years after application	Steevens *et al.* (1972)
DSMA	112 kg/ha	clay loam	No leaching below 15 cm with 51 cm of water.	Dickens and Hiltbold (1967)
DSMA	112 kg/ha	loamy sand	52% moved 23 cm deep with 51 cm of water.	Dickens and Hiltbold (1967)
DSMA	9 kg/ha	sandy loam	No leaching below 30 cm with 580 cm water.	Johnson and Hiltbold (1969)
MSMA	9 kg/ha	sandy loam	No leaching below 30 cm with 580 cm water.	Johnson and Hiltbold (1969)
DSMA	18-36 kg/ha	sandy loam	25% leached below 30 cm deep. None detected at 90 cm.	Johnson and Hiltbold (1969)
MSMA	18-36 kg/ha	sandy loam	75% leached below 30 cm deep. None detected at 90 cm.	Johnson and Hiltbold (1969)
MSMA	40 kg/ha	silt loam, fine sandy loam and loamy sand	No leaching in any soil below 30 cm over 6 years.	Hiltbold *et al.* (1974)
MSMA	NG	silt loam, silty clay loam and loam soil from forest floors	No leaching in any soils.	Norris (1974)
Cacodylic acid	NG	silt loam, silty clay loam and loam soil from forest floors	84% leached 8 cm with 86 cm water in silt loam and 50% in loam.	Norris (1974)

[a] NG = not given.

[b] All soil concentrations assumed to be for total arsenic.

4.3.5.3 Phosphate Fertilizers

Both natural and synthetic phosphate fertilizers contain significant quantities of arsenic as an impurity. Senesi et al. (1979) examined 32 commercial fertilizers for concentrations of arsenic, bismuth, lithium, and selenium. Arsenic levels ranging from 2 mg/kg (calcium cyanamide) to 321 mg/kg (triple superphosphate) were detected in all of the samples, and arsenic was generally present in greater amounts than any other trace element. The presence of As in synthetic nitrogen fertilizers was attributed to impurities in H_2SO_4, HNO_3, catalysts, equipment corrosion, coaters and conditioners used to prevent caking, and materials such as gypsum and limestone, added to form a granular product (Senesi et al. 1979).

The amount of arsenic entering the soil annually through use of phosphate fertilizers can be estimated. Assuming an application rate of 84 kg of phosphorous per hectare (Tisdale and Nelson 1968) and an arsenic concentration of 4 mg/kg in the fertilizer, approximately 0.3 g of arsenic is added per hectare of soil. Application of fertilizer with arsenic levels of 300 mg/kg would add 25 g of arsenic per acre.

Despite this evidence of possible arsenic contamination of soils via fertilizer additions, Goodroad and Caldwell (1979) found that a clay loam and silt loam fertilized with concentrated superphosphate and a mixture of phosphate fertilizers, for 19 years did not accumulate arsenic as compared with control plots. For the clay loam, increased As levels were not observed to a depth of 47.5 cm; for the silt loam, a slight decrease was observed in the C horizon (primarily mineral layer deep in soil) due to leaching of $CaCo_3$ to the C horizon with subsequent transfer of arsenic adsorption sites out of the higher horizons. Despite this evidence, at typical rates of fertilizer application, arsenic levels in all of these fertilizers will probably add to soil contamination over years of application and accumulations in animals fed these fertilizers.

4.3.5.4 Plant Uptake

Arsenic occurs in soil in the vicinity of geological deposits and from mining and smelting operations primarily via deposition of As-contaminated particulates. Also arsenic in the form of organic arsenicals[1] and sodium arsenite used as herbicide provides another pathway of exposure. Plants accumulate arsenic either through root uptake from soil solution, or, via leaf absorption of aerially-deposited arsenic.

Some fraction of the accumulated inorganic arsenic may be transformed to organic pentavalent arsenic (NAS 1977). Since information on the fate of the inorganic form is limited, no general conclusions can be made. More is known about organic arsenicals. Organic arsenic may be complexed to plant constituents and remain fairly stable (NAS 1977);

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[1] Primarily monosodium methanearsonate (MSMA), disodium methanearsonate (DSMA) and cacodylic acid.

a minor fraction was reported to metabolize and volatilize from leaves in coastal bermudagrass (Duble et al. 1969). In general, for numerous plant species and several organic forms of arsenic, studies with C^{14} markers indicated little metabolism, but translocation of arsenic from the point of entry to other parts of the plant (NAS 1977, see Table 4-2). Levels toxic to plants can therefore accumulate.

Typical and maximum concentrations of arsenic in edible plants have been compiled in various sources (e.g., NAS 1977, U.S. EPA 1980) and are discussed in greater detail in Section 5.2. In general, however, natural background levels may range from 0.01 mg/kg (dry weight) to as high as 3 mg/kg. Arsenic levels in plants grown in treated soil (with lead arsenate, arsenate and arsenite salts, trioxide and organic arsenicals) range from trace levels to approximately 1,700 mg/kg (highest in tomatoes and lemon tree roots), with the majority of levels between 0.2 mg/kg to 10 mg/kg (NAS 1977: see Appendix A). Little information was available on concentrations in grass treated with arsenical turf improvers. Concentrations of 2 mg/kg were measured in grass grown on soil treated to levels of up to 550 mg/kg (Williams and Whetstone 1949).

Variability among plants is attributed to species differences, various application methods, different sampling times, the influence of soil characteristics on uptake, and other factors. At an orchard site (treated 10 years previously) the highest arsenic levels were found in onion tops at 2 mg/kg grown in soil with 233 mg/kg. Levels in cotton at 9 mg/kg were reported in soil (at 28 mg/kg arsenic previously treated with MSMA) (Johnson and Hiltbold 1969). Therefore, total arsenic level in soil by itself is not a reliable indicator of potential arsenic levels in plants. Kenyon et al. (1979) investigated the availability of arsenic to plants grown in soil that had received lead and calcium arsenate insecticides for years. The results indicated that the arsenic in the insecticide-treated soil (22.4 mg/kg as opposed to 1.9 mg/kg in the control) was tightly bound and did not translocate into any of the plant species grown. The factors determining solubility of arsenic in soil (discussed previously) are expected to determine actual plant uptake. The pentavalent form would be the prevalent soluble form in the aerobic layer in which plant roots grow. Certain agricultural practices such as liming and phosphorus application may lead to solubilization of bound arsenic and plant uptake.

4.3.5.5 Application of Arsenical Herbicides to Aquatic Systems

The total amount of arsenic used as an aquatic herbicide is small on a national scale but its use may have a significant impact on a particular system. An example of the propensity of aquatic systems to accumulate and store arsenic in the sediment layer was illustrated in a study of a lake receiving sodium arsenite herbicide applications (Lambou and Lim 1970). A Wisconsin lake had received 218,000 lb of arsenite, which was estimated to be distributed at 87 lb/acre or 380 mg/kg in the top 1-inch of sediment. Measured levels were not too different, ranging from 10 to 82 mg/kg with a mean of 49 mg/kg. The remainder of the arsenic (undetected) may have migrated deeper into the sediment or been methylated and released to the atmosphere. Other studies have indicated depletion of sediment-bound arsenic over time (Lawrence 1957), probably due to biological activity.

The sediment core profiles of lakes treated with sodium arsenite herbicides all showed sediment surface layer accumulation of arsenic, but no obvious correlation with iron, manganese, calcium or organic carbon content of the lake sediments (Kobayashi and Lee 1978) as might be expected from observations in soil. In sediment surfaces high in organic matter, arsenic was easily leachable with distilled water (60% of the total arsenic present) whereas in highly calcareous sediments, the arsenic was more tightly bound (only 25% of total As leached). In anoxic sediments, 34-49% of the total As was extractable by distilled water.

MacKenthun (1970) measured bottom sediment levels of sodium arsenate ranging from 10 mg/kg to 82 mg/kg after treatment of a pond at 2.6 mg/l. Ten months after treatment of a pond at 2.6 mg/l, no arsenic was detected in water; sediment levels were not reported (Dupree 1970). However, one year following treatment of small farm ponds which had been consecutively drained and refilled several times, sodium arsenite levels of 380 μg/kg were measured in bottom mud and 300 μg/l in water. This indicated long-term persistence of arsenic in sediment. Other chemical forms of arsenic were not reported so it is not known whether or not some of the arsenic initially in trivalent salt form was converted to other forms.

4.3.5.6 Summary

A significant amount of arsenic is used in agricultural applications as an insecticide and herbicide and is applied inadvertently as an impurity in fertilizers. Some arsenic is also used as a herbicide in aquatic systems. Arsenical pesticides include inorganic forms such as sodium arsenite and arsenic acid and organic forms such MSMA, DSMA and cacodylic acid. During pesticide application, a small amount of arsenic may be lost or transferred through drift. On reaching soil, the chemistry of all arsenicals is similar regardless of the form; adsorption by soil colloids and complexation and/or chelation are the most important immobilization processes. Soils high in clay content, organic matter, and available iron and aluminum have the greatest capacity for retaining arsenic. Leaching of all forms of arsenic has been observed in a variety of soil types. Movement appears to be confined to the top 30 cm in most soils but migration to a depth of 90 cm was observed in sandy loam. Transfer of arsenic from soil to surface water may occur in surface runoff. Microbial transformations of organic arsenicals to arsenate and arsenicals to methylated forms may be significant in agricultural soils. Methylated arsenic forms are highly volatile and can be lost from the soil to the atmosphere.

Phosphate and other mineral fertilizers of both natural and synthetic origin may contain significant quantities of arsenic as an impurity. Long-term use of highly-contaminated fertilizers is likely to result in the accumulation of high arsenic levels in soil.

Plants accumulate arsenic from natural background levels in soil as well as from agricultural soils treated with arsenic. Crops grown on treated soils generally have arsenic residues at levels less than 10 mg/kg; a maximum accumulation of 1,700 mg/kg was reported in tomatoes and lemon trees. The level of soluble arsenic in soil is a better indication of the potential for plant uptake than is the level of total arsenic.

Most of the sodium arsenite applied as a herbicide to aquatic systems migrates into the sediment layer and binds to inorganic and organic material. Some of the sediment held arsenic is eventually released to the water column due to microbial methylation.

4.4 DISTRIBUTION OF ARSENIC IN THE ENVIRONMENT

This section presents information concerning actual concentrations of arsenic detected in environmental media. A literature review and STORET were the main sources of monitoring data for arsenic in aquatic systems. Observations concerning arsenic concentrations contained in the STORET systems are discussed for ambient and effluent waters, sediment, dissolved and suspended matter. Published monitoring data concerning arsenic concentrations are presented for natural water, sediment, soil, the atmosphere and terrestrial plants. Environmental media of significance to a particular pathway or to human exposure are presented elsewhere. For example, fish tissue levels are discussed in Pathway 3 (4.3.3) and levels in drinking water and food are reported in the human exposure section (5.2). Whenever possible, the arsenic chemical forms present were identified; however, information of this detail was uncommon.

4.4.1 Natural Waters

For the nation as a whole, concentrations of total arsenic in natural waters are relatively low. Forty-six percent of the arsenic concentrations recorded in the STORET system do not exceed 1 μg/l; 91% are no higher than 10 μg/l. Table 4-12 presents the distribution of unremarked observations from 1975 through 1979 for major river basins and the United States. Regional differences among the major river basins are slight; the distribution for each basin is almost identical to the national distribution. The exception is the Great Basin, for which 17% of the concentrations are at or below 1 μg/l, and 64% are no higher than 10 μg/l.

TABLE 4-12. DISTRIBUTION OF TOTAL ARSENIC CONCENTRATIONS IN AMBIENT WATER FOR MAJOR RIVER BASINS AND THE UNITED STATES--UNREMARKED DATA IN STORET, 1975-1979

Major River Basin	Total No. of Observations	% Distribution of Observations at Concentration (μg/1)						
		≤0.001	0.002-0.1	0.2-1.0	1.1-10	10.1-100	100.1-1000	≥1000
Northeast	1472	30	0	39	27	3	1	<1
North Atlantic	1325	24	<1	35	34	5	1	<1
Southeast	2537	22	0	43	33	1	<1	<1
Tennessee River	879	8	<1	49	39	4	<1	0
Ohio River	3757	24	<1	22	50	4	<1	0
Lake Erie	1563	4	<1	26	68	2	<1	0
Upper Mississippi	6490	53	<1	8	34	5	<1	<1
Lake Michigan	823	35	2	25	31	7	1	0
Missouri River	3637	21	<1	12	52	13	2	1
Lower Mississippi	5291	15	0	13	61	11	<1	<1
Colorado River	2809	44	<1	9	33	10	2	<1
Western Gulf	1126	17	<1	13	52	17	1	0
Pacific Northwest	2301	18	<1	28	43	10	1	<1
California	1272	26	0	10	55	8	1	0
Great Basin	653	3	<1	14	47	33	2	0
Lake Huron	154	9	0	15	70	6	0	0
Lake Superior	205	12	0	47	40	0	0	0
Hudson Bay	14	7	0	14	79	0	0	0
UNITED STATES	36308	27(9800)	<1(121)	19(7046)	45(16285)	8(2781)	1(2781)	<1(51)

Note: ( ) = number of observations in concentration range.

Source: U.S. EPA STORET Water Quality Control Information Sytem, as of November 13, 1980.

TABLE 4-13. TOTAL ARSENIC CONCENTRATIONS DETECTED IN FRESHWATER

Sample	Arsenic Level	Reference
From 150 U.S. rivers	detectable in 7% of 1,500 samples	Kopp and Knower (1970)
From U.S. rivers and lakes	detectable in 21% of 727 samples	Krum *et al*. (1971)
Lakes in Wisconsin	2 to 56 μg/1	Chamberlain and Shapiro (1969)
U.S. rivers and lakes	10 to 1100 μg/1	Durum *et al*. (1971)
Columbia River	1.6 μg/1 - average	Wedepohl (1969)
River in industrial area (Taiwan)	0.21 mg As/1	Tseng *et al*. (1968)
Kansas River	2 to 8 μg/1	Angino et al. (1970)
Spring waters, high in bicarbonate (worldwide)	400 to 1,300 μg/1	Schraeder and Balassa (1966)

TABLE 4-14. TOTAL ARSENIC CONCENTRATIONS DETECTED IN SALTWATER

Sample	Arsenic Level	Reference
Pacific coastal water	3 to 6 μg/l	Wedepohl (1969)
Northwest Pacific	0.15 to 2.5 μg/l 1.2 μg/l - average	Wedepohol (1969)
Indian Ocean	1.3 to 2.3 μg/l 1.6 μg/l - average	Wedepohl (1969)
Southwest Indian Ocean	1.4 to 5.0 μg/l 3.0 μg/l - average	Wedepohl (1969)

Other miscellaneous observations of total arsenic levels in freshwater systems are reported in Table 4-13. Most values are compatible with those in the STORET data base. Exceptions include industrial areas and saline water bodies.

In addition to this information, STORET retrievals were made for the minor river basins comprising the Pacific Northwest and Missouri River major river basins for the purpose of examining concentrations in the vicinity of known arsenic sources and to determine the origin of unusually high arsenic levels. The results are discussed in the aquatic exposure section (6.2).

According to STORET data, total arsenic concentrations measured from ocean sampling in the Northeast, the Western Gulf, and California range from 0.7 μg/l to 220 μg/l. Sampling of arsenic in estuaries in eight regions revealed concentrations ranging up to 1,700 μg/l; the average among the regions is 3.15 μg/l. Some miscellaneous observations in saltwater are also reported in Table 4-14.

Sampling of arsenic in USGS monitoring wells has been limited to specific regions (U.S. EPA 1980c). Major river basins for which enough observations have been recorded to indicate well water quality are the Southeast, Ohio River, Lake Erie, Pacific Northwest, Tennessee River, and Missouri River. For the five-year period 1974-1979, maximum, minimum, and mean concentrations for these six basins are shown in Table 4-15.

TABLE 4-15. CONCENTRATION OF ARSENIC IN WELLWATER IN SIX MAJOR U.S. RIVER BASINS--STORET, 1975-1979

River Basin	No. Observations	Concentrations (μg/l) Maximum	Minimum	Mean
Southeast	104[a]	63	10	21.5
Tennessee River	125[a]	100	10	15.3
Ohio River	917	390	0	8.6
Lake Erie	353	500	0	11.1
Missouri River	330[a]	10	5	8.3
Pacific Northwest	93	14	0	1.9

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[a] Remarked data.

Source: U.S. EPA (1980c).

In the Californian Mojave Desert at China Lake and Boron, potable ground water was reported to contain arsenic (total) at concentrations of 8 μg/l to 33 μg/l (Whitnack and Marlens 1971). The analytical technique used was single-sweep polagraphy confirmed by x-ray fluorescence, which is superior to the previously commonly employed colorimetric silver diethyldithiocarbamate (SDDC) method. The method employed did not, however, distinguish between the trivalent and pentavalent forms of arsenic.

4.4.2 Effluent Waters

STORET monitoring data concerning arsenic in effluent waters indicate concentrations ranging up to 2,900 μg/l. The limited number of observations reflect local conditions within the river basins. In the areas known to receive arsenic discharges, average concentrations of total arsenic in surface water range from 1 μg/l to 418 μg/l. The overall average concentration in effluent is 56.8 μg/l. Table 4-16 displays sampling results for arsenic in effluent waters recorded in STORET.

4.4.3 Sediment

Concentrations of arsenic observed in sediment samples in STORET are generally two to three orders of magnitude higher than ambient water concentrations (U.S. EPA 1980). From 1975 to 1979, average concentrations for arsenic in sediment ranged from 3 mg/kg to 17 mg/kg dry weight, compared with concentrations of 9 μg/l to 42 μg/l in ambient water nationwide.

The mean concentrations from year to year over the 5-year period in each major river basin indicate that:

- average concentrations for arsenic in sediment are generally around 5 mg/kg for most regions of the nation; and that

- elevated concentrations from year to year occur in six major basins--North Atlantic, Ohio River, Upper Mississippi, Missouri River, Lower Mississippi and Pacific Northwest. The annual average concentrations in these six areas exceed 8 mg/kg.

Forstner (1976) reported arsenic levels (total) in sediment core samples from lakes; data are presented in Table 4-17. The maximum levels were always measured in the upper layer of the sediment, two to 25 times the levels measured in the deeper layers. The author attributed this to recent deposition of contaminated sediment on top of older, deeper sediment with lower background levels of arsenic.

One of the objectives in a study conducted by Kent and Johnson (1979) was the measurement of concentrations of arsenic in fish, water, and sediment of American Falls Reservoir, Idaho, a presumably uncontaminated water body. Though arsenic was detected in water and sediment,

TABLE 4-16. ARSENIC CONCENTRATIONS IN EFFLUENT WATERS -- UNREMARKED DATA IN STORET (1975-1979)

Major River Basin	No Observations	Arsenic Concentration (ug/1) Range	Mean
Northeast	23	3 - 100	15.7
North Atlantic	4	10 - 56	28.5
Southeast	282	0.1 - 217	41.8
Tennessee River	5	2.8 - 100	30.3
Ohio River	72	0 - 700	23.6
Lake Erie	4	1 - 10	5.8
Upper Mississippi	30	1 - 2900	517.9
Lake Michigan	6	1 - 2	1.2
Missouri River	5	0.5 - 70	14.5
Lower Mississippi	20	3.6 - 22	7.8
Colorado River	5	1 - 34	8.6
Western Gulf	5	11 - 20	15.8
Pacific Northwest	94	0.4 - 1203	18.5
California	1	6.0	
Lake Huron	1	1.0	
Hudson Bay	2	1 - 1	1.0
TOTAL FOR REPORTED BASINS	559	0 - 2900	56.8

Source: STORET Water Quality Control Information System, as of October 27, 1980.

it was not detected in fish. Concentrations of arsenic in water samples ranged from 1.5 μg/l to 33 μg/l and in sediment samples 1.36 mg/kg to 2.40 mg/kg. Table 4-18 presents analyses from sediment and water samples collected from four areas of the reservoir.

TABLE 4-17. ARSENIC CONCENTRATIONS IN SEDIMENT CORE SAMPLES FROM VARIOUS U.S. LAKES

	Concentration of Arsenic (mg/kg)		
Location	Background (Deep) Layer	Maximum Value (Upper) Layer	Reference
Lake Michigan	11	22	Ruch et al. (1970)
Lake Monona	2	51	Syers et al. (1973)
Lake Washington	10	200	Barnes et al. (1973)
Lake Erie	0.6	3.2	Walters et al. (1974)

4.4.4 Dissolved and Suspended Matters

Table 4-19 displays arsenic concentrations, dissolved and in suspended matter, as reported from STORET. The higher concentrations of arsenic in dissolved form appears in three of the six basins mentioned previously as having more accumulated arsenic in the sediment: the North Atlantic, Missouri River, and Pacific Northwest. Higher arsenic concentrations are also present in sediment in the Great Basin, as is the case for concentrations in ambient water. According to STORET, the highest arsenic concentrations in suspended matter appear in the Missouri River and Colorado River basins.

4.4.5 Soil and Plants

Arsenic is present in various types of minerals and ores. Tables 4-20 and 4-21 present concentrations in different inorganic mineral material. Natural background levels of arsenic in soil are usually less than 10 mg/kg (Sandberg and Allen 1975). In a survey of 60 soils uncontaminated by human activity, 30% had concentrations less than 5 mg/kg, 50% between 5 mg/kg and 10 mg/kg, and 20% greater than 10 mg/kg (Schroeder and Balassa 1966). Generally, sandy soils had lower levels and arid soils had higher levels than the average.

In a survey of 500 samples in Alabama taken from soil previously treated with calcium arsenate, arsenic levels had a similar frequency distribution as in the uncontaminated samples, with only 4% exceeding 10 mg/kg (Hiltbold 1968). Soils from orchards treated with arsenicals (lead arsenate) tend to have higher arsenic levels than other soils.

TABLE 4-18. ARSENIC RESIDUES IN WATER AND SEDIMENT AMERICAN FALLS RESERVOIR, IDAHO, 1974

	Arsenic Concentration			
	Water (µg/l)		Sediment (mg/kg)	
Location	Mean	Range	Mean	Range
Area 2	12.60	1.50 - 33.0	2.02	1.57 - 2.40
Area 3	15.67	3.0 - 25.5	1.83	1.38 - 2.20
Area 4	16.50	3.5 - 30.5	1.56	1.40 - 1.75
Area 5	4.67	3.0 - 8.0	1.38	1.36 - 2.05

Source: Kent and Johnson (1979).

TABLE 4-19. AMBIENT ARSENIC CONCENTRATIONS IN DISSOLVED AND SUSPENDED MATTERS, UNREMARKED DATA IN STORET, 1975-1979

	Concentration (μg/1)					
	Dissolved			Suspended		
Major River Basin	Observations	Maximum	Mean	Observations	Maximum	Mean
Northeast	493	8.0	0.7	338	11.0	0.4
North Atlantic	249	323.0	5.2	123	29.0	1.8
Southeast	1654	11.0	1.2	940	17.0	0.6
Tennessee River	235	6.0	1.0	61	3.0	0.4
Ohio River	854	53.0	0.9	354	17.0	0.9
Lake Erie	124	21.0	1.6	78	5.0	0.7
Upper Mississippi	595	17.8	3.0	343	31.0	1.0
Lake Michigan	213	4.0	1.0	117	7.0	0.4
Missouri River	1608	619.9	5.9	783	22000.0	46.0
Lower Mississippi	1846	150.0	1.9	1386	150.0	2.1
Colorado River	1300	58.0	3.1	517	1300.0	13.4
Western Gulf	699	59.0	3.2	246	46.0	2.0
Pacific Northwest	1238	2600.0	6.9	696	100.0	0.6
California	627	35.0	1.9	387	220.0	1.4
Great Basin	844	330.0	12.5	248	42.0	1.9
Lake Huron	151	8.0	0.9	46	4.0	0.8
Lake Superior	130	15.0	0.9	70	2.0	0.4
Hudson Bay	41	55.0	4.1	--	--	--
UNITED STATES	12901	2600.0	2.6	6733	22000.0	7.4

Source: STORET Water Quality Control Information System, as of November 13, 1980.

TABLE 4-20. CONCENTRATIONS OF ARSENIC DETECTED IN SOILS, ROCKS AND ORES

Sample	Arsenic Concentrations	Reference
Crystalline rock	2 mg/kg (average)	Bowen (1966)
Soils	unpolluted: 1-40 mg/kg 5 mg/kg (average) near smelter: 150 mg/kg	Schroeder and Balassa (1966) Miesch and Huffman (1972)
Shale	13 mg/kg (average)	Schroeder and Balassa (1966)
Igneous rock	2 mg/kg	Onishi and Sandell (1955)
Shale and deep sea sediments	10 mg/kg	Onishi and Sandell (1955)
Sandstone and limestone	1.5 mg/kg	Onishi and Sandell (1955)

TABLE 4-21. CONCENTRATIONS OF ARSENIC DETECTED IN COAL

Coal	No. Samples	Range of Conc. (mg/kg)	Arithmetic Mean (mg/kg)
Illinois[a]	113	1-120	14
Appalachian[a]	23	2-100	25
Western[a]	29	0.3-9.8	2.3
Eastern[b]	617	0.12-950	18
Combined[c] (82% IL)	101	0.5-93	14

Maximum = 950 mg/kg; weighted mean = 17 mg/kg

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[a] Gluskoter *et al*. (1977).
[b] Zuboric *et al*. (1979).
[c] Ruch et al. (1974).

Average concentrations in orchard soils have been reported from 165 mg/kg (58 samples) to 627 mg/kg (10 samples), with a maximum of 2,500 mg/kg (PAX 1973). Arsenical pesticide use in orchards essentially ceased in 1948 with the advent of DDT; the fact that the previously mentioned concentrations were published in 1971 indicated significant persistence of arsenic in soil.

A number of sites contaminated with arsenic from mine and smelter waste were investigated by Porter and Peterson (1977) in southwest England. Plant and soil samples were taken from two major areas that account for roughly 97% of the arsenic output in the area. Heavily contaminated sites were largely barren and supported a restricted number of plant species. Surviving plant species developed a tolerance to high concentrations of arsenic in the soil, and accumulated arsenic to high levels as shown in Table 4-22, levels significantly higher (by 4 orders of magnitude) than plants growing in uncontaminated areas.

Concentrations of arsenic in soil samples from the contaminated sites are exhibited in Table 4-23. In an analysis of the oxidation states of arsenic in several water-soluble extracts, arsenate was the major form characterized, with only small quantities of arsenite present. The water-soluble extract was assumed to be fairly representative of the fraction available for plant uptake and movement in the soil. The nitric acid extract, on the other hand, is representative of the total amount of arsenic present in the soil, bound and unbound. The oxidation states of arsenic were also analyzed in samples collected at depths ranging from the surface layer to 28 cm (Table 4-24). The highest levels were detected at depths greater than 15 cm indicating vertical movement of the element in both arsenate and arsenite form. Dimethylarsine was only detected in the upper 2 cm probably due to concentration of microbial activity there and the high volatility of the compound.

Kenyon et al. (1979) monitored vegetable crops planted on orchard land near Interlaken, NY, which had received applications of arsenic compounds for many years, and on nonorchard soil near Ithaca, NY. Concentrations of arsenic registered in edible portions of one of the five plants seeded, tomato 0.2 mg/kg dry weight on both orchard and nonorchard lands. Only arsenic concentrations in the soils exhibited the differences between the two locales: 1.9 mg/kg dry weight in nonorchard soil, 22.4 mg/kg in orchard soil.

4.4.6 Air

The average annual atmospheric concentration of arsenic in remote areas is 0.4 ng/m^3 compared with up to 0.03 μg/m^3 in the vicinity of smelters (NASN 1964). The annual average for all sites is 0.003 μg/m^3. Table 4-25 presents ambient arsenic levels and concentrations measured in the vicinity of specific sources. Levels associated with industries were generally two orders of magnitude greater than ambient levels and the highest levels (for human exposure) were reported near cotton gins and copper smelters. In concentrations emitted from heat-fired

TABLE 4-22. CONCENTRATIONS OF ARSENIC IN THE FOLIAGE OF VARIOUS PLANT SPECIES

	Concentrations (ug/g dry weight)	
Species	Maximum	Mean
Jasione montana	6640	2040
Calluna vulgaris	4130	1260
Agrostis tenuis	3470	1480
Agrostis stolonifera	1350	720
Calluna vulgaris	0.33	0.3
Agrostis tenuis	0.28	0.23

Source: Porter and Peterson (1977).

TABLE 4-23. CONCENTRATIONS OF ARSENIC IN SOIL SAMPLES FROM SITES CONTAMINATED WITH MINE AND SMELTER WASTE

Arsenic Concentration (μg/g dry weight)

Soil Samples Extracted with Concentrated Nitric Acid		% of Water Soluble		
Total Arsenic	Water Soluble	As(V)	As(III)	Other
26,530	1.18	78.0	10.2	11.8
24,200				
21,430	0.87	84.9	4.5	10.6
11,040	1.40	93.7	6.3	
9,590				
8,510				
20	0.16	79.2	20.8	

Source: Porter and Peterson (1977).

TABLE 4-24. CONCENTRATION AND SPECIES OF AVAILABLE ARSENIC IN WATER-SOLUBLE AVAILABLE EXTRACTS OF SOIL SAMPLES FROM SITES CONTAMINATED WITH MINE AND SMELTER WASTE

Sample	Concentrations of Arsenic (ug/g dry weight)		
Depth (cm)	As (III)	As (V)	As(CH$_3$)2
0 - 2	0.195	0.110	0.026
2 - 4	0.185	0.290	Not Detected
4 - 6	0.218	0.207	Not Detected
6 - 8	0.235	0.655	Not Detected
8 - 10	0.178	0.340	Not Detected
10 - 15	0.654	0.846	Not Detected
15 - 20	0.720	74.40	Not Detected
20 - 25	0.215	21.88	Not Detected
25 - 28	0.190	28.75	Not Detected

Source: Porter and Peterson (1977).

TABLE 4-25. ARSENIC CONCENTRATIONS DETECTED IN THE ATMOSPHERE

Sample Site	Concentration ($\mu g/m^3$) Range	Concentration ($\mu g/m^3$) Average	Reference
National urban ambient levels	0.001[a] - 0.083	0.003	Suta (1980)
Urban areas containing smelters		0.03	Suta (1980)
Remote areas		0.0004	Suta (1980)
Coal-fired power plants		0.003	Suta (1980)
Copper smelters[b]		≤0.99	Suta (1980)
Lead smelters[b]		≤0.29	Suta (1980)
Zinc smelters[b]		≤0.29	Suta (1980)
Cotton gins[b]		usually ≤0.59 but as high as 5.9 for very small subpopulation	Suta (1980)
Pesticide manufacturers[b]		≤0.0009	Suta (1980)
Glass manufacturers[b]		≤0.29	Suta (1980)

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[a] Detection limit 0.001 $\mu g/m^3$.

[b] These numbers have been selected as concentrations far enough away from the point of emission to expose at least a small subpopulation (≤230 people). The assumptions on which these levels are based are quite complex and should be reviewed before these numbers are used for other purposes. See the Suta (1980) document for a detailed discussion of the derivation of these numbers.

sources, the trivalent form is prevalent (see Chapter 4.3, Pathway 1). However, sampling indicates the presence of both the trivalent and pentavalent forms in other areas (Mushak et al. 1980).

4.5 OVERVIEW

The purpose of this section is to attempt an integrated overview of the significance of arsenic releases to and intermedia transfers between air, soil and surface water within the perspective of their potential contribution to water-borne routes of exposure. The most important pathways are emphasized based upon the magnitude of pollutant levels involved, the presence of toxicologically important chemical forms of arsenic, and the significance of the receiving medium as it relates to exposure of receptor populations. The following discussion is based on information presented in Chapters 3.0 and 4.0.

4.5.1 Important Fate Processes

Arsenic is subject to numerous chemical and biological transformations in surface waters and soil. Table 4-26 summarizes the most important of these processes. As previously noted, atmospheric transformations of arsenic are not considered but only transfers from the atmosphere to soil or water.

4.5.2 Transfer from Air to Surface Water and Soil

The majority of the arsenic initially released to the atmosphere is expected to be transferred eventually through rainout or fallout to water or land. The extent of dispersion depends upon the size of the particulate in the emission and the prevailing climatic conditions. Larger diameter particulates (>20 μm) tend to be deposited within 1 km of the point source. For smaller sized particulates, a mean residence time of 9 days was estimated, and this suggests a potential for transport of the adsorbed arsenic over a considerable distance before settling out. This is especially important for arsenic released from coal combustion processes because of the affinity of the element in its vapor form for smaller diameter particulates. Arsenic released from cotton gins is primarily associated with large diameter particulates, which would most likely be deposited near to the source. The relationship between arsenic levels and particulate size emitted by the remaining significant sources of atmospheric releases, such as smelting operations, is not known, however, the range in particulate size is expected to be great. Monitoring data indicate high levels of arsenic accumulation in the surface layer of soil immediately surrounding smelters which may be due to the contribution of larger-diameter particulate fallout.

Atmospheric emissions of arsenic may, therefore, have a significant impact on residue accumulation in local terrestrial and aquatic environments. An accumulation rate of approximately 3 mg of arsenic per m^2 of soil per year was estimated in the vicinity of a coal-fired power plant and the rate likely to be higher in the vicinity of other

TABLE 4-26. CHEMICAL AND BIOLOGIC TRANSFORMATION OF ARSENICALS IN SURFACE WATER AND IN SOIL[a]

Reactions and Changes	Relative Rate of Change	Products	Biologic Activity	Probability of Occurrence	Conditions for Occurrence	Further Possible Changes
1. Salt formation	High	Insoluble arsenic salts	Insoluble and inactive	Very high	Presence of iron, aluminum, calcium, and magnesium in soil or sediment.	Formation of an arsenic analogue of fluoroapatite, an extremely insoluble complex mineral.
2. Adsorption	High	Soil- or sediment-arsenic complex	Fixed and inactive	High	Fine soil and sediment colloidal and organic matters.	Formation of sediment in aquatic systems.
3. Ion exchange	High	Soil- or sediment-arsenic complex	Fixed and inactive	High	Soil or sediment with high exchange capacity.	Exchange release by other salts to react further as 1 or 2.
4. Demethylation	Low	Inorganic o-arsenic acid	Reacts as 1, 2, and 3	Low	Microorganisms for demethylation (aerobic)	Same as 1, 2, and 3.
5. Reduction	Low	Arsines	Very active	Low	Aerobic and anaerobic conditions or specific microorganisms.	React rapidly to form pentavalent arsenicals; then same as 1, 2, and 3.
6. Oxidation[b]	Moderate	Pentavalent arsenicals	Reacts as 1, 2, and 3	High	Aerobic soil or surface water conditions.	Same as 1, 2, and 3.
7. Methylation	Low	Methylarsines	Very active	Low	Presence of specific bacterial microroganisms (anaerobic and aerobic).	React rapidly to form pentavalent arsenicals; then same as 1, 2, and 3.

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[a] Adapted from NAS (1977).

[b] Refers to oxidation of arsenic from trivalent to pentavalent.

sources. Soil levels near atmospheric sources are on the order of two to three orders of magnitude greater than background levels. Approximately 10% of the annual arsenic loading to the Puget Sound was attributed to atmospheric fallout.

The chemical form of arsenic initially released in combustion-associated atmospheric emissions is usually arsenic trioxide. The trioxide readily is converted to arsenite on contact with water, however, so this is the form usually entering soil and water. Some arsenic is also released from mining and milling in the same form as it is found in ores. Cotton ginning processes presumably release arsenic in the form of arsenic acid.

4.5.3 Releases to Land and Transfer to Ground Water

Releases of arsenic in solid waste disposal to land account for the largest fraction of total environmental releases, approximately 65% excluding pesticide application to land. Due to long-term persistence of arsenic wastes in soil and the potential for mobilization of arsenic under certain environmental conditions, these releases can sometimes result in significant exposure problems.

From most sources, the predominant form released is expected to be arsenite. The initial form is most important in the period of time before an equilibrium is established, according to the conditions of the receiving medium. After equilibrium is achieved, the prevailing environmental conditions have a significant influence on arsenic's specification and, therefore, on its potential for mobility, retention, biotransformation and volatilization. The importance of the initial form appears to be somewhat overshadowed by the site conditions following establishment of equilibrium.

The method of application or disposal onto land is an important factor in the potential for migration of arsenic into groundwater. Arsenic in slag or sludge from tailing's ponds is often disposed of at managed disposal sites. The percentage of disposal sites lacking the proper precautions to prevent runoff or leaching and migration of toxic substances (such as clay or polyethylene liners, tiling, etc.) must be known in order to assess the significance of these wastes as a source of arsenic pollution on a national scale. There may also be a potential for leaching of arsenic from slag used as road construction material. Fly ash is usually disposed of, following settling, in ponds which are eventually filled solid. Protective lining of these ponds is a relatively recent phenomenon. The arsenic concentrations in fly ash are sometimes high, and the surface area to volume ratio greater than in mining and smelting wastes; therefore the potential for migration from fly ash ponds may be considerable. Municipal or industrial landfills are often reported to contribute to elevated concentrations of arsenic in ground water.

Following release to land, arsenic in the upper aerobic soil layers will be present as arsenate and under reduced conditions (saturated or the deeper layer of soil) as arsenite. Soil types in which arsenate will be mobile include soils low in iron and aluminum oxides, kaolinite (around neutral pH) rather than montmorillonite clay soil, high pH, loamy and sandy soils. Soil types in which arsenite will be mobile include all of the above except the presence of low pH, which will release bound arsenic from Fe, and increases mobility. Organic matter content does not appear to be an important factor in retention of arsenic. Under very reduced conditions (i.e., in bogs), arsine gas may be formed. In the majority of industrial landfills, conditions are not expected to be favorable for microbial activity so than biological transformation of arsenic forms (oxidation, reduction, methylation) is not expected to be as significant as in agricultural soils. This would mean that loss from soil through volatilization of methylated arsenic compounds would be minimal.

The national distribution of land disposal of arsenic solid wastes and of environmental parameters influencing mobility are discussed in Section 5.2.2.1. Appendix D includes maps and tables depicting or describing the distribution of these sources or factors in the U.S.

4.5.4 Discharges to Surface Water and Waste Water Treatment

Industrial discharges of arsenic-containing effluents to surface water contribute a minor fraction (~1%) of the total intentional releases of the element. Aquatic input from pesticide runoff and phosphorus detergent use contribute, at most, another 1% of the total releases. Discharges from POTWs and urban runoff (nonpoint source) contribute the largest fraction of the aquatic discharges. The reason that POTWs discharge more arsenic than industrial operations may be due to the use of tertiary treatment in industry. Primary and secondary processes are inconsistent and often inefficient (<50% removal) in removing arsenic from waste streams while tertiary treatment processes usually achieve a removal efficiency of 85% or higher. It is important to note that since arsenic is an indestructable element, an increased efficiency in water treatment results in an increase in releases to land with the disposal of sludge. There does not appear to be as much of an effort spent to reclaim and recycle arsenic in sludges as for certain other metals.

The aquatic chemistry of arsenic is determined by pH, Eh, cations such as Ca and Ba, and the clay and iron hydroxide content of the sediments. In well oxidized waters, species of H_3AsO_4 predominate. In more reducing waters, species of H_3AsO_3 appear as well as arsenic sulfides. Iron and aluminum oxides adsorb and complex with arsenic, and settle to the sediments. The sediment layer acts as sink for arsenic, which is bound by clay, iron and aluminum oxides and sulfides. This substantially reduces concentrations in the water column. Organic sediment material has not been identified as a major sink for arsenic. Within the sediments, arsenic may be solubilized by methylating bacteria, reduced to the gas, arsine, and/or desorbed from reduced iron oxides as As(III). Elements such as Be, Cu, and Cr may also act to control dissolved concentrations of arsenic in the water column.

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5.0 EFFECTS AND EXPOSURE--HUMANS

5.1 HUMAN TOXICITY

5.1.1 Introduction

Based on a number of clinical, occupational and epidemiological studies, arsenic is generally considered to be a human carcinogen. The lack of suitable animal models to demonstrate carcinogenic activity or other toxic effects, however, has proved to be a major obstacle in elucidating dose-response relationships for the various arsenicals. This section of the report deals primarily with the effects of water-soluble forms of arsenic, each of which has different toxicological properties on laboratory animals, and human studies that reflect dose-response relationships; human epidemiologic data are presented separately under subsection 5.1.4 of this section.

5.1.2 Metabolism and Bioaccumulation

5.1.2.1 Absorption

The absorption, bioaccumulation, metabolism and excretion of arsenic compounds are variable, depending on the particular form of the compound (i.e., organic vs. inorganic, pentavalent vs. trivalent, etc.), it's water solubility, lipid solubility, and the route of administration. Thus, difficulties are often encountered in clearly delineating the exact uptake and absorption of different arsenic compounds by various routes of exposure (U.S. EPA 1980). Arsenic is absorbed into the human body primarily via ingestion or inhalation, and, to a lesser extent, via dermal contact.

Oral

Absorption via the digestive tract and mucous membranes is heavily dependent upon solubility and physical properties of the compound. Soluble salts of arsenic are absorbed from all mucous membranes and parenteral sites of administration much more readily than the oxide (Klaasen 1980, Harvey 1975). Finely divided powders are absorbed more readily from the gastrointestinal tract and are more toxic than coarse particles since the latter may be eliminated in the feces before they are dissolved (Klaasen 1980, Stewart and Stolman 1960). Pentavalent inorganic arsenicals are absorbed from the digestive tract more readily than the trivalent forms, possibly because they react less with the intestinal contents and mucosa. Organic trivalent forms are not readily absorbed from the digestive tract for the same reason; however, these forms may be partially degraded in the intestinal tract and the resulting inorganic arsenic is more readily absorbed. The pentavalent organic forms are variously absorbed, but none are absorbed completely (Harvey 1970, Klaasen 1980).

Coulson et al. (1935), Ray-Bettley and O'Shea (1975), Crecelius (1977a, b) and Charbonneau et al. (1978a) report that in humans, greater than 95% of an ingested dose of soluble trivalent arsenic and greater than 80% of an ingested dose of arsenic compounds in certain seafoods are absorbed from the gastrointestinal tract, with less than 5% of the dose being found in the feces. Mappes (1977) reports daily urinary excretion of 69-72% of a daily oral intake by a human subject of approximately 0.8 mg of trivalent arsenic. Crecelius (1977a) reports excretion within 61 hours of 80% of a total dose of 63 μg of pentavalent inorganic arsenic following ingestion of the dose in a wine sample. However, insoluble arsenic triselenide was found to pass through the gastrointestinal tract with very little absorption (Mappes 1977).

Studies with laboratory animals have resulted in findings similar to those in the human studies as concerns absorption of arsenic compounds. In rats, 88% absorption from the gastrointestinal tract was noted for arsenic trioxide solution and 70-90% for arsenate solution (Coulson et al. 1935, Urakabo et al. 1975, Dutkiewicz 1977); 90% and 98% of an arsenic trioxide solution was absorbed in pigs (Munroe et al. 1974) and monkeys (Charbonneau et al. 1978a), respectively. When Atlantic grey sole containing arsenic (1 mg fish/kg b.w.) was fed to adult female monkeys, pigs and adolescent monkeys, absorption from the gastrointestinal tract was 90%, 70% and 50%, respectively (Charbonneau et al. 1978a). Only 40% and 30% absorption was observed following oral administration of arsenic trioxide suspension to rabbits and rats, respectively (Ariyoshi and Ikeda 1974).

Dermal

Trivalent arsenicals are generally more readily absorbed through the skin as compared with pentavalent forms (Harvey 1975). Dermal absorption of inorganic arsenic is increased when applied as a lipid-soluble ointment (Dustin 1933). Similarly, Patty (1948) reports increased dermal absorption of arsenic when epidermal lesions are present compared with absorption from normal skin. Dutkiewicz (1977) reports an uptake rate of arsenate via the rat tail as high as 33.1 μg/cm^2/h with exposure to concentrations up to 0.2 M (~15 g/l); extrapolation of these results suggests a rate of uptake of arsenate through human hands of 23.2 mg/hour.

5.1.2.2 Distribution and Bioaccumulation

Within 24 hours after oral or parenteral administration of inorganic arsenite to humans, monkeys, rabbits, dogs and guinea pigs, most of the absorbed dose is found cleared from the blood and distributed to solid tissue, with highest accumulations in the liver, kidney, spleen, lungs and walls of the gastrointestinal tract. Smaller levels are found in the brain, heart, uterus, muscle or remaining in the blood. Arsenic in the blood of these species is bound in nearly equal amounts to the blood cells and proteins in the serum. Approximately 2 weeks after exposure, high accumulation of arsenic may be found in the skin, hair and nails, due to reaction of the arsenic with the sulfhydryl groups as in keratin, arsenic may remain fixed at these sites for years. Deposition and long-term retention of the compound may also occur in the bone. No notable levels of arsenic are found in the spinal fluid of humans (Klaasen 1980, Hunter 1942, Harvey 1975).

Several studies have indicated transplacental passage of arsenic in humans with subsequent accumulation in the fetus. Analysis of fetal organs 11 hours after delivery from a mother who took As_2O_3 approximately 12 hours before delivery, indicated accumulations of 0.74 mg As_2O_3/100 ml in the liver, 0.15 mg/100 ml in kidney and 0.021 mg/100 ml in brain (Curry 1972). Kagey et al. (1977) found cord blood levels to approximate those of mothers in 101 maternal-newborn tissue sets. In a presumably healthy Japanese population, Kadowaki (1960) found measurable levels of arsenic in fetuses at least by the fourth month of gestation and increasing to the seventh month. The highest levels were found in the brain tissue, as well as the liver and skin. These data could suggest vulnerability of the human fetal nervous system to arsenic exposure, since the metal generally crosses the blood-brain barrier in relatively small amounts. However, limitations of this study include the lack of specification as to the chemical nature of the tissue arsenic and the lack of information concerning dietary history of the population (U.S. EPA 1980).

The rat appears to demonstrate an exception to the pattern of clearance of arsenic from the blood and subsequent distribution of the metal to solid tissue, as observed in humans and various other mammals studied. Following subcutaneous injection of radiolabelled potassium arsenite to rats, a uniquely high amount of the label is retained in the blood (95-99% in the red blood cells, bound to the globin of hemoglobin). These concentrations in the blood indicate a two- to eight-fold increase over those that would be expected with uniform distribution throughout the body, with concentrations in solid tissue being much lower than expected (Hunter et al. 1942). The half-time of blood clearance of both pentavalent and trivalent inorganic arsenic in the rat is approximately 60-90 days (Lanz et al. 1950, Ariyoshi and Ideda 1974). This rate of clearance is notably higher than the half-times of 1, 5 and 35 hours of the three-compartment model of clearance for humans and dogs, as proposed by Charbonneau et al. (1978b). Similarly, Overby and Frederickson (1963) calculated half-times of approximately 6 hours for the rapid phase, slightly longer for the second phase, and approximately 60 hours for a slow phase in a three-compartment model for blood arsenic clearance in several mammalian species (excluding the rat; exact species not stated in secondary reference).

Although the blood is the main vehicle of transport of arsenic compounds, biliary transport has also been reported for a number of species. The bile excreted arsenic is reabsorbed. Klassen (1974) reports a higher biliary excretion rate in rats as compared with rabbits or dogs and Cikrt and Benko (1974) noted a higher biliary excretion rate for trivalent arsenic forms than for pentavalent in the rat (approximately 10:1).

5.1.2.3 Excretion

The primary route of excretion of arsenic in man and animals is in the urine; only a few percent of an absorbed dose is excreted in the feces. Other minor routes of elimination include the skin, hair, nails, and sweat (Hunter et al. 1942, Ducoff et al. 1948, Mealey et al. 1959, Kadowaki 1960 , Lander et al. 1965).

The rate of urinary excretion is dependent upon animal species, chemical form of the arsenic, and the route of administration. Pentavalent arsenicals are generally excreted rapidly from the body via the kidneys, while the trivalent forms tend to bind to sulfhydryl groups of tissue proteins and are slowly excreted as they are released (Webb 1966). Organic arsenicals are excreted more rapidly than inorganic forms. The general order of excretion rates is as follows (in the reverse order of toxicity): RAs-X > As^{+5} > As^{+3} > AsH_3 (Fowler 1977). The slow excretion of the trivalent forms is the basis for their greater toxicity; the pentavalent forms are so rapidly excreted that very little accumulates unless very large doses are administered over a long time period (Klaasen 1980).

A greater proportion of arsenite is excreted from humans following parenteral administration as compared with oral ingestion. Urinary excretion begins 2-8 hours following injection of arsenite, but up to 10 days may be required for complete elimination of a single dose and up to 70 days after repeated administration (Hunter et al. 1942, Harvey 1970, Klaasen 1980). Mealey et al. (1959) report three phases of urinary excretion in man after a single intravenous dose of radiolabelled arsenite (2.3 mCi/70 kg) with half-lives of 2 hours, 8 hours and 8 days, respectively; more than 99% of the dose is excreted within the first 15 hours, excretion occurs at a low constant rate over the next 156 hours and the remainder is removed at a much lower rate. Crecelius (1975, 1977a, b) reports half-lives of 10 hours and 30 hours for the inorganic trivalent form and methylated forms of arsenic, respectively, when ingested in wine. Following ingestion of wine containing 50 μg As(III) and 13 μg AS(V), approximately 80% of the dose was measured in the urine after 61 hours. Crecelius (1977b) reports a biological half-life of less than 20 hours for seafood arsenic, while Coulson et al. (1935) and Westöö and Rydälv (1972) report elimination of 70-80% of an ingested dose of organic seafood arsenic within 2 days. Mappes (1977) reports maximum urinary excretion by 3 hours following a single ingested dose of arsenite solution, with approximately 25% of the dose appearing in the urine 24 hours post-exposure. Successive daily intakes of 0.8 mg arsenite resulted in daily urinary clearance of approximately 66% of the daily intake, after 5 days.

The urinary excretion of arsenic in rats is much slower than in other mammalian species, as would be expected considering that 80-90% of the metal is fixed in the hemoglobin of the red cell, which must break down before the arsenic can be released (NAS 1977). Ducoff et al. (1948) found urinary excretion in the rat to be less than 10% during the first 48 hours following injection of 47 μCi ^{74}As-labelled sodium arsenite as compared with approximately 70% in rabbits and 30-45% in man 48 hours after injection of 235 μCi and 3.0 mCi or 2.7 mCi, respectively. Less than 10% of the excreted arsenic was found in the feces of all three species. Arsenic levels in the rat blood remained higher for up to 96 hours. Fowler (1979) reports a half-life of arsenic in rats of approximately 60 days after a single exposure, while Urakabo et al. (1975) reported 84 days. (Chemical form of arsenic and route of administration not stated in either report.)

In humans, conversion from arsenite to arsenate in the body is favored by the in vivo redox equilibrium. The low toxicity and high recovery of pentavalent arsenicals in the urine and excreta indicate little occurrence of the reverse biotransformation (Klaasen 1980). Both arsenate and arsenite can be converted to organic arsenic compounds such as methylarsenates (Federal Register 1978). The major urinary excretory product in both exposed and unexposed humans is dimethyl-arsinic acid; other forms of arsenic in the urine include arsenite, arsenate, methylarsonic acid, and other organically bound arsenic compounds (Vallee et al. 1960, Buchanan 1962, Schroeder and Balassa 1966, Frost 1976, Lisella et al. 1972, Vallee 1973, Peoples 1975).

Crecelius (1977a) reports excretion within 61 hours of 63 μg arsenic consisting of 50% dimethylarsenic acid, 14% monomethyl arsenic, and 8% each the two inorganic forms, following ingestion of a wine sample containing 50 μg trivalent arsenic and 13 μg pentavalent arsenic. Increased levels of pentavalent arsenic and dimethylarsenic and near background levels of trivalent arsenic were measured in the urine of a subject in the same study following ingestion of well water containing 200 μg arsenate (quantitative data not available in secondary reference). Organic arsenic ingested in seafood appeared not to be biotransformed in the body, but was excreted intact in the urine; chemical treatment of this product released dimethylarsenic.

Mealey et al. (1959) report levels of "pentavalent" arsenic ranging from about 60% after 1 day to 80% with further time following parenteral administration of As^{74} trivalent arsenic. This "pentavalent" excretion product probably was a mixture of cacodylic, methyl arsenic, and inorganic pentavalent arsenic; the latter was at levels greater than any contaminating levels in the original trivalent dose, indicating the conversion to arsenate and methylated forms in the body.

Oral administration of arsenite or arsenate to both dogs and cows resulted in conversion of both arsenic forms to methylated arsenic. No quantitative distinction was made between inorganic arsenic forms (Lakso and Peoples 1975).

Following intravenous administration to dogs of radiolabelled arsenic acid, inorganic arsenic was detected as the major form in plasma up to about 2 hours post-dosing. Levels of the dimethyl arsenic could be detected as early as 10 minutes post-dosing, and by 6 hours approximately 90% plasma arsenic was in this form, with little detection of the monomethyl forms. Dimethyl arsenic was the predominant form in the urine from days 1-6 (Tam *et al*. 1978, 1979). Charbonneau *et al*. (1978b) detected approximately 80% of an As^{74}-labelled arsenic acid dose lodged in the red cells following intravenous administration to dogs; in time, the arsenic content was distributed between cells and plasma. Dimethyl arsenic was detected in the cells by 10 minutes post-dosing, total conversion was apparent by 6 hours and dimethyl arsenic was detected in the urine by 1 hour post-dosing.

Detection of dimethyl arsenic in the urine, feces, and blood of adult male rats fed ferric methanearsonate suggested methylation of monomethyl arsenic *in vivo*. The dimethyl arsenic was the predominant form in the blood, but was detected in minor amounts in the urine and feces, a finding possibly indicating selective retention of dimethyl arsenic by rat erythrocytes (Odanaka et al. 1978). The two above studies indicate involvement of the erythrocyte and the liver in biosynthesis and transport during dimethyl arsenic formation in the rat and dog (U.S. EPA 1980).

Conversion of trivalent inorganic arsenic to pentavalent inorganic arsenic *in vivo* has been reported in dogs after intravenous infusion of arsenite; both inorganic forms were detected in the plasma, urine, and glomerular filtrate. In the dog, arsenate appears to be resorbed by the proximal renal tubule, reduced and excreted as arsenite; the reverse oxidation also occurs (Ginsburg 1965). Benko et al. (1976) detected virtually no trivalent arsenic in the urine of mice when As^{74}-labelled arsenite was administered after 18 days of pre-exposure to a large dietary level (250 mg/l) in drinking water. The relative amount of pentavalent arsenic formed was dependent on the time lapse between pre-treatment and dosing with the arsenite (this pentavalent form may, in fact, have been dimethyl arsenic acid). Winkler (1962) detected mainly arsenate in the livers of rats fed arsenite.

Data supporting the *in vivo* conversion of the pentavalent arsenic to the trivalent form is less conclusive due to the analytical methods used (U.S. EPA 1980). Similarly data concerning dimethylation of methylated arsenic *in vivo* are limited, although the available information appears to indicate the absence of such conversions (U.S. EPA 1980).

5.1.2.4 Summary

In summation, water-soluble arsenicals are readily absorbed by ingestion, inhalation, and dermal contact; some nonpolar organic forms are also absorbed from the GI tract and skin. Arsenic trioxide is not well absorbed. Pentavalent arsenic (both organic and inorganic) is better absorbed than trivalent arsenic, presumably due to less interaction with membranes of the gut. In general, arsenites are better absorbed through the skin than arsenates, with the rate of absorption highly dependent upon the lipid solubility of the compound.

Little is known about the kinetics and metabolism of arsenic and its compounds, but it appears that much of a given dose is excreted in urine. In humans exposed to trivalent or pentavalent arsenic, elimination occurs predominantly as methyl arsenic and dimethyl arsinic acid in urine. However, neither of these metabolites was detected in urine following ingestion of organoarsenicals in seafood, although total urinary arsenic was markedly elevated, a finding suggesting organoarsenicals may be excreted without metabolic degradation.

5.1.3. Human and Animal Studies

5.1.3.1. Carcinogenicity

Epidemiological evidence suggests an association between cancer incidence and the ingestion of drinking water containing higher than normal concentrations of arsenic, including people in Cordoba Province in Argentina (Bergoglio 1964) and Taiwan (Tseng et al. 1968). (These studies are discussed in Section 5.2 of this chapter.) In most cases, however, human exposures to arsenic have been concurrent with exposures to other agents, thus one cannot exclude the possibility that other cofactors may have played a role in the carcinogenic response.

There are no animal-models for study of arsenic-induced cancer. Animal studies have failed to demonstrate carcinogenicity for arsenic compounds, even when administered at maximum tolerated doses for the animal's lifespan.

Byron et al. (1967) observed no carcinogenic effects in Osborne-Mendel rats following dietary administration of either sodium arsenite (15-250 mg/kg dietary concentrations) or sodium arsenate (30-400 mg/kg for 2 years). Survival was reduced slightly by the arsenate and both compounds caused weight reduction. At the highest doses (250 mg/kg arsenite and 400 mg/kg arsenate), a marked enlargement of the common bile duct was evident. Similarly, no carcinogenic effects were observed in a 2 year feeding study involving administration of sodium arsenite or sodium arsenate to beagle dogs at dietary levels of 5, 25, 50 or 125 mg/kg of either compound. However, data from this study are inadequate for risk extrapolation due to the poor survival of the test animals in the arsenite-treated group and the insufficient length of treatment and observation periods (Byron et al. 1967).

Fairhall and Miller (1941) reported no carcinogenic effects associated with feeding rats lead or calcium arsenate at daily doses of 10 mg daily for up to 2 years. A similar response was observed by Kroes et al. (1974) in specific-pathogen-free Wistar rats fed either 1850 mg/kg lead arsenate or 463 mg/kg sodium arsenate in their diets.

In another study, no carcinogenic effects were observed in rats exposed from weaning to senescence to sodium arsenite at levels of 5 mg/l in drinking water. Elevated levels of serum cholesterol and lower levels of blood glucose were noted in male rats (Schroeder et al. 1968).

Hueper and Payne (1962) also reported no development of skin, lung, or liver cancer in rats or 50 C57BL mice exposed to arsenic trioxide in the drinking water with or without 12% ethanol. The concentration of arsenic trioxide was increased from 4 mg/l to 34 mg/l over the first 15 months; administration was continued until 24 months for a daily intake of 0.2-0.8 mg/arsenic/rat.

Frost et al. (1962) reported no carcinogenic effects in chicken, pigs, or rats following long-term administration of arsanilic acid. No adverse effects were seen in chickens or pigs fed the compound for 4 years (dose not stated in secondary reference) or in pigs fed 0.01% arsanilic acid for three generations. When male and female weanling rats from the F_2 generation of a six-generation study (in which 0.01% or 0.05% arsanilic acid was fed) were held on a 0.01% arsanilic acid diet for 116 weeks, the overall tumor incidence was the same for all groups and was similar to the historical incidence of tumors in the colony (35-45%).

Levels of 10 mg/l sodium arsenite administered in the drinking water of virgin female C3H/St mice for 15 months reduced the incidence of spontaneous mammary tumors from 82% to 27%, but caused significant enhancement of the growth rate of spontaneous or transplanted mammary tumors (Schrauzer and Ishmael 1974).

The sole positive response is a report by Osswald and Goerttler (1971) who found a considerable increase in the incidence of leukemia in Swiss mice, both mothers and offspring, after daily subcutaneous injections of 0.5 mg/kg sodium arsenate administered during pregnancy for a total of 20 injections followed by a 2-year observation period. Lymphocytic leukemia occurred within 24 months in 46% (11/24) of the mothers, 21% (7/34) of the male offspring and 16% (6/37) of the female offspring. When groups of the offspring from the arsenic-treated-females were given an additional 20 subcutaneous injections of 0.5 mg/kg sodium arsenate at weekly intervals, 41% (17/41) of the males and 48% (24/50) of the females developed leukemia compared with 9% (3/35) males and 0% (0/20) females in the control groups. Similarly, administration of 20 weekly intravenous injections of 0.3 mg/kg sodium arsenate to female mice caused lymphoma in 58% (11/19) of the treated animals compared with 0% (0/16) in female controls.

5.1.3.2 Teratogenicity

Little information is available on teratogenic effects associated with oral administration of arsenic compounds. Hood and coworkers (1977) reported that intraperitoneal administration of sodium arsenate (40 mg/kg) to mice during gestation had a considerably greater effect on prenatal mortality, reduction of fetal weights, and occurrence of fetal malformations than did oral administration of this compound (120 mg/kg) according to the same treatment regimen.

James (1966) observed no terata in offspring of four ewes fed 0.5 mg potassium arsenate/ kg b.w. throughout most of their pregnancy.

Intravenous or intraperitoneal injections of sodium arsenate during gestation have been shown to induce developmental malformations in hamsters, mice, rats, and chick embryos (Beaudoin 1974, Ferm et al. 1971, Ferm 1977, Franke et al. 1936, Hood and Bishop 1972, Hood et al. 1977, Ridgeway and Karnofsky 1952). Other than chick embryo studies, the lowest injected dose tested to date (15 mg sodium arsenate/kg injected intravenously or intraperitoneally on day 8 of gestation) induced teratogenic effects in exposed hamsters (Ferm et al. 1971, Ferm 1977).

The incidences of fetal deaths, resorptions, and developmental abnormalities observed in offspring of golden hamsters injected intravenously on day 8 of gestation with 15, 17.5 or 20 mg/kg b.w. sodium arsenate were found to be dose-dependent. Litter resorptions ranged from 5% to 80%; the incidence of malformations ranged from 20% to 90%. Malformations included anencephaly (over 80% of the embryos), rib malformations (65%), exencephaly (30%), malformations of the genitourinary system (20%), cleft lip and palate and renal agenesis. A no-observed-effect-dose has not been established for this species (Ferm et al. 1971).

Similar responses have been found in Swiss Webster mice injected intraperitoneally on day 9 of gestation with 45 mg sodium arsenate/kg maternal body weight; i.e., fetal malformations, increased fetal resorptions, and significantly decreased fetal body weights compared with controls (Hood and Bishop 1972). Some 60% of 96 implantations were resorbed or dead and 63% or the offspring were grossly malformed. Defects included cleft lip, fused vertebrae, agnathia, and exencephaly.

Hood et al. (1977) also compared the teratogenic responses of sodium arsenate and sodium arsenite in mice. They found that intraperitoneal injection of 10 mg sodium arsenite/kg on one of days 7-12 produced effects comparable to those noted subsequent to an intraperitoneal injection of 45 mg/kg sodium arsenate.

Effects similar to those observed in hamsters and mice have also been reported for the rat. Beaudoin (1974) noted teratogenic effects in Wistar rats following intraperitoneal injection of 20-50 mg/kg b.w. sodium arsenate on days 7-12 of gestation. Maximal effects occurred with the 30-mg/kg dose injected on either day 8, 9, or 10. The types of malformations were comparable to those noted in hamsters and mice.

Ridgeway and Karnofsky (1952) detected no specific gross abnormalities in chick embryos exposed to 0.2 mg/kg sodium arsenate during embryogenesis. However, this test procedure is insufficiently reliable for an assessment of safety in species such as man that possess chorioallantoic placentae.

Thus, animal studies have demonstrated that an intravenous or intraperitoneal injection of 15-50 mg/kg b.w. sodium arsenate during gestation is capable of inducing developmental malformations in several species. Few oral studies are available. A single study conducted with mice suggests a reduced response by the oral route. Arsenite appears to be more toxic to the embryo than arsenate, but once again, comparative values are available for only one species by one route of administration.

5.1.3.3 Mutagenicity

Few mutagen-detecting assays that reliably predict mutagenic effects in man have been conducted for arsenic compounds. Most available mutagenicity data are focused on *in vitro* cytogenetic analysis of sodium arsenate. No host-mediated or dominant lethal tests are available (NAS 1977).

Beckman and associates (1977) found a significantly higher ($P<0.001$) incidence of chromosomal aberrations in leukocytes taken from arsenic-exposed smelter workers (8.7%) when compared with controls (1.3%). However, due to simultaneous exposure to other agents, the effects cannot be attributed to arsenic with certainty. A total of 819 and 1012 mitoses were examined for the arsenic exposed and control groups, respectively. The three predominant aberrations found were gaps (6.8% vs. 0.9% in controls); chromosome aberrations including dicentric chromosomes, rings, and acentric fragments (2.3% vs. 0.09% in controls); and chromatid aberrations (o.5% vs. 0.3% in controls).

Petres and Hundeiker (1968) and Petres *et al*. (1970, 1972) have reported chromosomal breakage in human lymphocytes taken from individuals after long-term *in vivo* exposure to arsenical compounds, as well as after short-term exposure to sodium arsenate in culture. *In vivo* studies with phytohemagglutin-stimulated lymphocyte cultures from 13 individuals who had previously undergone intensive arsenic therapy (up to 20 years prior to this study) indicated an increased incidence of chromosomal aberrations compared with controls. Expressed as the frequency per 1000 mitoses, the incidence of secondary constrictions was 49; gaps, 51; "other" lesions, 26; broken chromosomes, 61; compared with 12, 7, 1, and 2 per 1000 mitoses, respectively, in the controls.

In separate experiments, Petres *et al*. (1970) found that exposure of human lymphocytes in culture to 0.1 μg/ml sodium arsenate resulted in pulverization of 33% of the metaphase plates; at concentrations of 2 μg/ml or greater, 80 to 100% of the metaphase plates were pulverized. In addition, the "mitosis index" and "[^{3}H] thymidine labelling index" were decreased.

Limarzi (1943) reported irregularly shaped nuclei and chromatin fragments in bone marrow samples of humans 4 days after treatment with Fowler's solution (KOAs=O).

The effects of sodium arsenate and arsenite on human lymphocytes and diploid fibroblasts in culture were examined by Paton and Allison (1972). The addition of sodium arsenite (0.29-1.8×10^{-8}M) to lymphocyte cultures for 48 hours prior to fixation produced chromatid breaks in 60% of 148 metaphases examined. With sodium arsenate, the highest non-toxic concentration (0.58×10^{-8}M) caused no notable increase in the number of breaks. With diploid fibroblasts, exposure to 0.29-5.8×10^{-8}M sodium arsenite for 24 hours prior to fixation caused chromatid breaks in 20% of the 459 metaphases examined.

Arsenate was found to increase the total frequency of exchange chromosomes in *Drosophila melanogaster* treated with selenocystine (Walker and Bradley 1969). The significance of this study is difficult to assess since many unrelated compounds cause similar effects.

Arsenic trichloride was found to enhance significantly viral transformation of Syrian embryo cells by a simian adenovirus, SA7. An enhancement ratio of 2.2 above controls was seen with a concentration of 0.003 mM $AsCl_3$ (Casto *et al*. 1979).

Arsenic compounds also induce mutagenic effects in bacteria. A net positive result was reported by Nishioka (1975) in a rec-assay with *Bacillus subtilis* H17 and M45 for several arsenic compounds. A more distinct rec-effect was seen with the sodium arsenite and arsenic trichloride than with sodium arsenate. Nishioka also observed an increased frequency of tryptophan reversions in several strains of *Escherichia coli* treated with 1.6×10^{-4}M sodium arsenite. Tryptophan reversions were induced most frequently in strain WP2uvrA (lacking DNA repair gene urvA), while strain CM_{571} (lacking the functional DNA repair gene RecA) showed little response to sodium arsenite. These results suggest that the RecA function may be essential for mutagenesis by sodium arsenite, while the uvrA gene has little effect (Nishioka 1975, Flessel 1977).

Rossman *et al*. (1975) found that sodium arsenite decreased the survival of ultraviolet-irradiated *E. coli* strain WP_2, but had no effect on the survival of RecA mutant WP10. These authors also suggested that arsenite may inhibit a RecA-dependent step in the repair of ultraviolet-induced DNA lesions.

Thus, chromosomal aberrations have been induced in human lymphocytes exposed in culture to sodium arsenate and arsenite. Chromosome breaks have also been found in lymphocytes taken from humans exposed to various arsenicals *in vivo*. *In vitro* cytogenetic analysis indicated that arsenite induced a higher incidence of aberrations in human lymphocytes than did arsenate. Bacterial studies also suggest more marked effects with the arsenite material.

5.1.3.4 Other Toxic Effects

Very little quantitative data are available regarding dose-response relationships for arsenic compounds. Most information concerns human occupational or epidemiologic exposures, which often do not allow for precise dose measurements, exposure periods or chemical speciation, and may be complicated by a variety of other contributing factors. Little information has been reported regarding animal models for toxic effects seen in arsenic-exposed humans.

In general, the toxicities of the various forms of arsenic are related to their rate of clearance from the body and, thus, to their degree of accumulation in the tissues. Tissues rich in oxidative systems (e.g., alimentary tract, kidneys, liver, lungs, epidermis) are particularly susceptible to arsenicals. The general order of toxicity is as follows: $AsH_3 > As^{+3} > As^{+5} > RAs - X$. The toxic action of pentavalent inorganic arsenic is thought to result from uncoupling of mitochondrial oxidative phosphorylation, which interferes with phosphate transport and phosphorylation. Presumably, the arsenate competes for inorganic phosphate, forming unstable arsenate esters (Klaasen 1980, Fowler 1977). The trivalent inorganic forms of arsenic appear to exert toxic effects via reaction with sulfhydryl enzyme systems essential to cellular metabolism indirectly interfering with their activity (Klaasen 1980). The trivalent inorganic arsenicals have also been known to interfere with active transport processes of potassium, sodium, hydrogen ion, monohydrogen phosphate, water, glucose, and certain amino acids (Webb 1966b).

In humans, the symptoms of acute arsenic exposure and the extent of these symptoms are dependent upon the type of arsenical, the amount of arsenic, the route of exposure, the age of the subject, as well as other variables. In general, the major characteristics of acute arsenic poisoning in humans are gastrointestinal disturbances and cardiac abnormalities. Subacute exposure results in nausea, vomiting, diarrhea, leg cramps; and disorders of the peripheral nervous, hematopoietic, cardiovascular, hepatic and integumentary systems (NAS 1977).

Peripheral neuropathy may begin any time from several days to several weeks after arsenic exposure and may persist for several years. With chronic or subacute arsenical intoxication, the neuritis is symmetrical, widespread and painful, affects both upper and lower extremities with both sensory and motor involvement (although motor involvement is rare). Histopathologic evidence has verified peripheral nerve damage, especially long-axon myelinated nerve damage (Heyman et al. 1956, Jenkins 1966, Chhuttani et al. 1967, Ohta 1970, LeQuesne and McLeod 1977). Initially, symptoms include tingling or numbness sensations in the soles of the feet and palms of the hand; these sensations may spread to proximal sites of the extremities. Symmetrical muscle weakness, severe pains in the limbs and crampy pain in the muscles, foot drop and wrist drop, diminished touch sensation, and absence or diminution of touch, pin-prick and temperature sensations (in more severe cases) are characteristic (Chhuttani and Chopra 1979, Fowler 1979, Kyle and Pease 1965, Heyman et al. 1956).

Although gross cardiac manifestations are rare, electrocardiogram abnormalities do occur with arsenic intoxication, including flattening and inversion of the T-wave and prolongation of the Q-T interval (Barry and Herndon 1962, Goldsmith and From 1980, Butzengeiger 1949, Glazener et al. 1968). Arsenic probably exerts a direct toxic effect on the myocardium, since these electrocardiogram alterations can not be correlated with changes in serum electrolytes (Fowler 1977). Jenkins (1966) reported sudden cardiac deaths following acute poisoning of the myocardium with or without associated brain stem medullary failure. In addition to severe myocardial injury, evidence has been reported of peripheral vascular disturbances, endo-angitis, gangrene of the extremities, atropic acrodermatitis and periorbital and ankle edema (Butzengeiger 1940, 1949, Limarzi 1943, Heyman et al. 1956, Kyle and Pease 1965). Blackfoot disease, a peripheral vascular disorder resulting in gangrene of the extremities has been positively associated with chronic arsenism (Tseng 1977) (see discussion in following subsection).

In six cases of arsenic intoxication, Kyle and Pease (1965) noted anemia, leukopenia, prominent basophilic stippling, and altered bone marrow erythropoiesis. The anemia was secondary to hemolysis and bone marrow depression. The anemia and leukopenia of acute arsenic poisoning appear to be reversible features (U.S. EPA 1980). A very prominent feature of arsine poisoning is hemolysis resulting in free methemoglobin and oxyhemoglobin in the plasma. In addition, there may be extensive bleeding from the gums, lips, and nose (Neuwirtova et al. 1961, Meuhrcke et al. 1963, Jenkins et al. 1965).

There is also evidence of liver and kidney disorders following arsenic poisoning. Frejaville et al. (1972) noted widespread intravascular coagulation and swollen liver and kidney mitochondria in several cases of arsenic intoxication. Cirrhosis and ascites have been observed following ingestion of Fowler's solution and in occupational arsenic exposures (Franklin et al. 1950, Butzengeiger 1940, 1949, Luchtrath 1972). St. Petery et al. (1970) noted mild abnormalities in liver function tests and indications of destructive influence on renal tubules, membranous ultrastructures, and enzyme systems, including proteinuria, glycosuria, and hematuria following ingestion of inorganic arsenic.

Renal failure, secondary to massive intravascular hemolysis is a prominent feature of arsine poisoning, but not of other arsenic compounds. The kidneys characteristically contain hemoglobin casts in the tubule lumens; cloudy swelling and necrosis of the proximal tubule cells, albuminuria, azotemia, and altered serum electrolyte concentrations have also been reported, all of which may continue for many months after termination of arsine exposure (Neuwirtova et al. 1961, Nielson 1968, Hocken and Bradshaw 1970, Uldall et al. 1970). The characteristic coppery skin pigmentation observed is thought to result from the presence of methemoglobin rather than jaundice, as measured bilirubin levels have been normal (Jenkins et al. 1965).

Other late-emerging symptoms of subacute or chronic arsenic exposure include skin lesions such as erythematous eruptions, melanosis, and other abnormal pigmentations and keratoses of the extremities, all of which are usually less apt to be reversible, as compared with other features of arsenic poisoning. Dry, falling hair, dry scalp, skin and brittle loose nails, and white striae in the fingernails (Mees lines) are characteristic signs of long-term exposure. Melanosis is most often seen on the upper and lower eyelids, around the temples, on the neck, on the areolae of the nipples and in the folds of the axillae, or, in more severe cases, on the abdomen, chest, back, and scrotum along with hyperkeratosis and warts. Depigmentation, especially on the pigmented areas and hyperkeratoses and exfoliation over the palms and soles are characteristic of chronic arsenic poisoning (Holland 1904, Reynolds 1901, Fowler 1979). Skin lesions may develop long after the exposure period, when concentrations of arsenic in the skin have returned to normal. Also, skin lesions, particularly hyperkeratosis, are thought to be related to skin cancer (Pinto and McGill 1953, Vallee et al. 1960, Buchanan 1962). A variety of studies have demonstrated that chronic arsenic exposure results in a characteristic sequence of skin effects: hyperpigmentation, hyperkeratosis and finally, skin cancer. Only a small proportion of the keratoses, however, evolve into skin cancer, and only after long periods of time (NAS 1977).

As stated previously, most data regarding human effects are based on occupational or epidemiological studies, with limited dose-response information. Several studies which give some quantitative data are briefly described in the following paragraphs.

5.1.4 Epidemiologic Studies

The literature on the epidemiology of incidents of arsenic-related effects is limited, primarily, to clinical reports of cases or groups of cases. Few controlled epidemiologic studies have been conducted, and even case reports are often lacking data on the form of arsenic and the level of exposure. The basic source of information for this review is the EPA Criteria Document for Arsenic (U.S. EPA 1980). Primary sources were also reviewed for additional detail. The following sections summarize reports of associations between exposure to waterborne arsenic and human health effects, as well as reports of health effects associated with the ingestion of foods or liquids contaminated with arsenic, because waterborne arsenic may be the cause of contamination. The case reports/studies are summarized in Table 5-1. In most studies, the chemical form of arsenic present was not specified and concentrations were generally reported as total arsenic.

5.1.4.1 Drinking Water Contamination

Several incidents have been reported of arsenic contamination of drinking water in Japan. Tsuchiya (1977) describes studies of 200 families living within a 7-km radius of an abandoned mine, which produced copper and arsenic trioxide. The highest arsenic concentration found in well water was 0.125 mg/l, while the concentration in most wells was 0.05 mg/l or less. Most of the persons identified as having arsenic poisoning had a history of occupational exposure to arsenic. One couple, however, without occupational associations, had consumed drinking water in the vicinity containing 0.125 mg/l arsenic. The wife was said to be suffering from Banti syndrome (congestive splenomegaly) while the husband was diagnosed as having liver cirrhosis. It was speculated that these diseases may have been related to arsenic ingestion. In another case, wastewater from a plant producing arsenic-sulfide contaminated local well water. About 60 cases of arsenic poisoning were identified among inhabitants residing within 400 m of the plant, where arsenic concentrations in well water were 0.05 mg/l and over with a maximum reported concentration of 3 mg/l.

Investigators in Antofagasta, Chile have studied the relationship between high arsenic levels in the municipal drinking water and various health effects including hyperkeratosis, bronchopulmonary disease, chronic cough, lip herpes, cardiovascular manifestations, chronic diarrhea and abdominal pain (Borgono and Greiber 1972, Borgono _et al_. 1977, Zaldivar 1974, 1976a). According to Zaldivar and Guillier (1977), the 265,000 inhabitants of Antofagasta Province represent the largest population at risk from arsenic in drinking water. The arsenic contamination results from the leaching by rainwater from strata of lava and volcanic sediments, which have a high arsenic content. The chemical form of arsenic present was not specified. Investigators observed a significantly higher prevalence of symptoms related to arsenic poisoning in Antofagasta as compared with other areas with lower arsenic levels. Furthermore, Zaldivar and Guillier (1977) and Borgono _et al_. (1977) describe the reductions in arsenic levels and symptom prevalence in the city of Antofagasto, which occurred following the installation of a municipal water treatment system. They report a weighted mean concentration of arsenic for the period 1955-1970 of 0.6 mg/l, which decreased to 0.08 mg/l for the period 1970-1972, a factor of 7.5. The incidence rate for chronic arsenical dermatosis in adults dropped by a factor of approximately 16 over the same time period. Borgono _et al_. (1977) report that children born after the installation of the water treatment system did not develop cutaneous lesions, which young children had developed in prior years. The authors point out that the absence of lesions may be due to an increased period of latency at lower doses and urge continuation of periodic examinations.

TABLE 5-1. EPIDEMIOLOGIC STUDIES OF THE HEALTH EFFECTS OF WATERBORNE ARSENIC EXPOSURE

Location	Route of Exposure	Level / Type of Exposure	Study Populations	Health Effects/ Results	Reference
Argentina, Cordoba Province	Drinking water	2.8-54.5 mg/l due to geologic contamination	Deaths in the province, skin cancer cases	Proportion of deaths due to cancer: 23.8% in arsenic area; 15.3% in non-arsenic area	Arguello *et al.* (1938) Bergoglio (1964) Lisella (1972)
Canada, Nova Scotia	Drinking water	Geological contaminations			Hindmarsch *et al.* (1977)
Chile Antofasasta	Drinking water	Geological contamination of water supplies up to 0.96 mg/l arsenic	Several population surveys	Estimated dose for skin cancer induction: 0.62-1.15 mg/day; latency 14-23 years	Zaldivar and Guillier (1977) Borgono *et al.* (1977)
Great Britain, Herefordshire	Drinking water	Arsenical pesticide back syphoned into water supply	Farm family	One fatality; six non-fatal poisonings	Tsuchiya (1979)
Nakajo	Drinking water	Wells contaminated with waste from arsenic sulfide plant; many wells 1-2 mg/l arsenic; maximum 3 mg/l	Case in the plant vicinity	60 cases of As poisoning identified from clinical observations	Terada (1960)
Shimane	Drinking water	Contamination of wells from mine producing copper and arsenic trioxide; most wells 0.05 mg/l or less; highest 0.125 mg/l; next highest 0.07 mg/l.	Examination of 200 families living within 7 km radius of the mine	7 cases of arsenic poisoning; 5 suspect cases	Tsuchiya (1977)

TABLE 5-1. EPIDEMIOLOGIC STUDIES OF THE HEALTH EFFECTS OF WATERBORNE ARSENIC EXPOSURE (continued)

Location	Route of Exposure	Level / Type of Exposure	Study Populations	Health Effects/ Results	Reference
Poland, Reichenstein, Silesia	Drinking water	12.2 mg/l, source and type of contamination unknown		Skin cancer	Lisella (1972) Geyer (1898)
Taiwan Peiman Yi-Chu Districts	Drinking water	Geological contamination of artesian wells, levels 0,01-1.82 mg/l arsenic, maybe predominantly trivalent, lysergic acid or related compounds present	Surveys of 37 villages with a population of 40,421	Skin cancer prevalence 10.6 per 1000 population. Positive correlations betweem arsenic levels in well water and skin cancer prevalence	Yeh (1963) Tseng (1968-1977) Lu (1975,1977a, b)
United States Oregon, Lane County	Drinking water	Geological contamination of water supplies; levels range 0-2.15 mg/l, mean 0.009 μg/l; presumably pentavalent	Skin cancer cases and controls	No positive associations	Morton *et al.* (1976)
Minnesota Perham	Drinking water	Contamination of wells by buried insecticide arsenic 12-21 g/liter	Clinical study of 13 persons exposed for 2.5 months	Intermittent GI symptoms	Feinglass (1973) U.S. EPA (1977)
California Lassen County	Drinking water	0,1-1,4 mg/l liter arsenic	Not specified	No specific illness at 1.4 g/liter	Goldsmith. *et al* (1972)
Japan, Okayama	Powdered milk	Sodium phosphate stabilizer with 6% As_2O_5; milk contained 21-34 μg arsenic per gram; doses range 2.5-4.6 mg.	Bottle-fed infants	12,131 cases of arsenical poisoning with 130 fatalities	
Ube	Soy Sauce	Arsenic contamination (90-100 μg/mL) from an unspecified source; exposure over 2-3 week period	About 400 persons exposed, 220 cases examined	Swollen liver and abnormal EKG in 80% of cases; symptoms disappeared 2 weeks after exposure ceased	Mizuta (1956)

A similar situation of drinking water contamination exists in the Cordoba Region of Argentina and in Nova Scotia, where geological conditions contribute to high arsenic levels (Arguello _et al_. 1938; Bergoglio 1964; and Hindmarsch _et al_. 1977). Bergoglio (1964) reports the results of a study in Argentina from 1949-1959, which found a higher proportion of cancer deaths in the arsenical region than in the rest of the Cordoba province: 23.8% vs. 15.3% (no dose information is available in the secondary source). Tseng _et al_. (1968) refer to the District of Reichenstein, Silesia, Poland where, in about 1889, an unusual cancer epidemic was associated with arsenic from mining slag heaps leaching into ground water. Apparently, the construction of a new water supply in 1928 eliminated the problem.

Some of the most intensive epidemiologic investigations of exposure to waterborne arsenic have occurred in Taiwan where a population of about 100,000 on the southwest coast is exposed to high (0.8-1.82 mg/l) concentrations of arsenic (chemical form not specified) in water from deep wells. Yeh (1963) conducted a general survey of six villages with a total population of 3,938 to determine the prevalence of chronic arsenical intoxication. Of the total population, 80.7% were examined and 36.8% of that population was found to have signs of chronic arsenical intoxication. The highest prevalence rates ranging from 93%-100% were found in the age groups over 50 years. The survey also identified 35 cases of skin cancer, which equal a total prevalence of 1.1%. For those over the age of 50, the prevalence was 6.8%.

Yeh (1963) also studied an endemic peripheral vascular disorder called Blackfoot disease, which results in gangrene of the extremities and reported close associations between this disease, signs of arsenical intoxication, and skin cancer. The associations, however are insufficient to establish casual relationships (U.S. EPA 1980a). For example, Lu _et al_. (1975, 1977a, b) analyzed water samples from areas with endemic Blackfoot disease and identified lysergic acid or a related compound. Since lysergic acid has well-known vasoconstrictive effects, it, rather than arsenic, could be the cause of Blackfoot disease. Tseng _et al_. (1968) reports on additional investigations in Taiwan where, by the end of 1965, 37 villages with a population of 40,421 had been surveyed. Within this population the overall prevalence rate of skin cancer was 10.6 per 1000 population; for hyperpigmentation the prevalence was 184 per 1000; for keratosis the prevalence was 71 per 1000; and for Blackfoot disease the prevalence was 9 per 1000. Arbitrary segregation of population groups into "high" (above 0.6 mg/l), "medium" (0.3-0.6 mg/l), and "low" (below 0.3 mg/l) arsenic exposure groups resulted in an obvious association between increasing concentrations and increasing prevalence of skin cancer.

The prevalence rate for skin cancer in the "high" arsenic area was 21.4 per 1000 population, while in the "low" exposure area the rate was 2.6 per 1000 population. In the age groups over 60 years, the corresponding rates for "high" and "low" exposure areas were 192 per 1000 population and 27.1 per 1000.

In contrast to the positive associations between arsenic contamination of drinking water and skin cancer found in Taiwan, Morton et al. (1976) did not find a similar relationship in Lane County, Oregon. There are several possible explanations for the discrepancy (U.S. EPA 1980a). First, the levels of arsenic were substantially lower in Oregon (mean: 0.009 mg/l; range 0-2.15 mg/l). Second, the trivalent form of arsenic present in the Taiwanese water samples was not prevalent in the Lane County samples (U.S. EPA 1980a). Other explanations include smaller sample size; differences in socioeconomic, racial, and dietary factors; and the possibility that other carcinogens were present in Taiwan as either casual or potentiating factors. A more detailed discussion of the relationship between arsenic in drinking water and skin cancer is found in Section 7.1.6.

5.1.4.2 Food contamination

Tsuchiya (1977) presents a review of various outbreaks of arsenic poisoning in Japan. The first incident, in 1955, resulted from the consumption of powdered milk, which included a sodium phosphate stabilizer contaminated with 6% As_2O_5. The concentration of pentavalent arsenic in the liquid milk was 21-34 μg/gram. Investigations identified 12,131 cases including 130 fatalities, primarily infants on bottle-feeding, Other effects reported included a variety of CNS-related disturbances, including hearing damage, abnormal brain wave patterns, increased incidence of mental retardation, epilepsy and other indications of brain damage, fever and abdominal swelling secondary to hepatomegaly, as well as, the usual coughing, rhinorrhea, conjuctivitis, vomiting, diarrhea and melanosis. The estimated exposure from ingestion was approximately 3.5 mg/day of arsenic resulting in total intake of 90-140 mg (Hamamoto 1955, Okamura et al. 1956, Yamashita et al. 1972, Masahiki and Hideyasu 1973).

Another case of food contamination was reported by Mizuta et al. (1956) who examined 200 Japanese patients who had ingested approximately 3 mg arsenic per day in contaminated soy sauce during a 2-3 week period. Facial edema and anorexia were noted in 85% of the subjects, exanthemata, desquamation and hyperpigmentation in about 10% and peripheral neuropathy in 20% of the exposed population. Headaches, fever, abnormal EKG's (but no evidence of congestive heart failure) and a few abnormalities in liver-function tests (but no severe degenerative changes) were also observed (Mizuta et al. 1956).

5.1.4.3 Medicinal Uses

Silver and Wainman (1952) reported abnormal pigmentation changes and gastrointestinal symptoms in humans some 13 months after ingestion of 8.8 mg arsenic trioxide (as Fowler's solution) daily for 28 months as a remedy for asthma. Redness and puffiness about the eyes and hyperkeratosis developed after approximately 1.5 years and neurological symptoms after 2 years. Hyperkeratosis persisted and a previously developed asthma worsened even after termination of exposure.

Fierz (1965) reported an incidence of hyperkeratosis of greater than 50% in patients administered more than 4 g arsenic trioxide in Fowler's solution for long periods of time some 6-25 years prior to the study. As little as 600 mg resulted in keratotic changes in one patient and 750 mg had been consumed by another with skin cancer; skin cancer was noted in 8% of the patients.

5.1.5 Overview

5.1.5.1 Ambient Water Quality Criterion--Human Health

The U.S. EPA (1980a) has established a zero ambient water concentration for the maximum protection of human health from the potential carcinogenic effects of arsenic exposure through ingestion of water and contaminated aquatic organisms. The water quality criterion is based on a number of epidemiologic studies that have shown that both ingestion and inhalation of arsenic increase the risk of cancer induction in human lung and skin tissues.

The concentration of arsenic in water calculated by the U.S. EPA (1980a) to keep any additional lifetime cancer risk below 10^{-5} is 0.022 µg/l based upon the data of Tseng et al. (1968) and a linear, non-threshold-dose response model for epidemiologic studies.

The National Primary Drinking Water Standard for arsenic is set at 50 µg/l for allowable arsenic concentrations in drinking water based on exposure, toxicity, technological and economic considerations (U.S. EPA 1980a); a variance guidance allows levels of up to 100 µg/l for up to one year until controls are in place. The discrepancy between the Criteria and Standards and Office of Drinking Water criteria is currently under study at EPA.

5.1.5.2 Additional Health Considerations

There is considerable evidence to demonstrate that arsenic has toxic potential for humans subsequent to environmental exposure via ingestion (see Table 5-2). Water-soluble arsenicals are readily abosrbed by ingestion, inhalation, and dermal contact. Few studies, however, have indicated the chemical species of exposure, thus complicating the understanding of this problem. Due to the large number of chemical forms of arsenic and the different toxicological properties of each, correlative evaluations are difficult to make. In general, arsines are the most toxic, followed in decreasing order of toxicity by arsenites, arsenoxides, arsenates, pentavalent arsenicals, arsonium compounds, and metallic arsenic.

TABLE 5-2. ADVERSE EFFECTS OF ARSENICALS ON MAMMALS[a]

Adverse Effect	Species	Lowest Reported Effect Level	No Apparent Effect Level
Carcinogenicity	Human	0.3 mg arsenicals in drinking water; epidemiological study	- - - - - - - - - -
	Rat	- - - - - - - - - -	463 mg/kg Na arsenate in diet for 2 years[c]
	Rat	- - - - - - - - - -	250 mg/kg Na arsenite in diet for 2 years[c]
	Rat	- - - - - - - - - -	1,850 mg/kg Pb arsenate in diet for 2 years[d]
	Rat	- - - - - - - - - -	10 mg/rat/day Ca arsenate in diet for 2 years
Teratogenicity	Ewe	- - - - - - - - - -	0.5 mg/kg K[b] arsenate in diet during gestation
	Mouse	120 mg/kg Na arsenate orally	- - - - - - - - - -
	Mouse	10 mg/kg Na arsenate intraperitoneally	- - - - - - - - - -
	Hamster	15 mg/kg Na arsenate intraperitoneally	- - - - - - - - - -
Mutagenicity			
Chromosome Aberrations	Human (lymphocytes in culture)	0.1 μg Na arsenate per ml medium	- - - - - - - - - -
Chromosome Breaks	Human (lymphocytes in culture)	0.29×10^{-8} M Na arsenate	- - - - - - - - - -
Chromosome Breaks	Human (lymphocytes in culture)	- - - - - - - - - -	0.58×10^{-8} M Na arsenate
CNS Damage	Human (infant)	3.5 mg arsenic per day in milk	- - - - - - - - - -
Peripheral Neuropathy	Human	3 mg/day Ca arsenate in soy sauce for 2-3 wks.	- - - - - - - - - -
Hyperkeratoses/ Hyperpigmentation	Human	8.8 mg arsenic trioxide orally for 28 mo.	- - - - - - - - - -

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[a] Data taken from Section 5.1.3 of this chapter.

[b] Equivalent to 23 mg/kg/day based on consumption of 15 g feed/day in 300-g rat.

[c] Equivalent to 12.5 mg/kg/day based upon the assumptions above.

[d] Equivalent to 92.5 mg/kg/day based on the assumptions above.

There is also considerable evidence for an association between arsenic and disease in humans. Skin lesions, including cancer and a circulatory disorder known as Blackfoot disease are major clinical problems associated with chronic arsenism. Animal models, however, have not demonstrated the carcinogenicity of arsenic, even when administered for long periods of time at near maximal tolerated concentrations. Clarification is needed as to whether arsenic is carcinogenic solely for man or whether some vital factor is lacking in animal models.

Animal studies have shown that arsenic causes fetal death at high doses and malformations at lower dosages in mice, rats, and hamsters. An intravenous or intraperitoneal injection of 15-50 mg/kg b.w. sodium arsenate during gestation is capable of inducing developmental malformations in several species. A single study conducted with mice suggests a reduced response to exposure by the oral route. In general, arsenite appears to be more toxic to the embryo than arsenate, but only few comparative studies have been done.

Chromosomal aberrations have been induced in human lymphocytes exposed in culture to either sodium arsenate or arsenite. Chromosome breaks have also been found in lymphocytes taken from humans exposed to various arsenicals *in vivo*.

Other toxic manifestations of arsenic exposure in man include effects on the gastrointestinal tract, cardiac abnormalities, peripheral neuropathy, kidney and liver disorders, and characteristic skin disorders (hyperpigmentation, hyperkeratosis).

5.2 HUMAN EXPOSURE

5.2.1 Introduction

As discussed in Chapter 4.0, arsenic is found in all environmental media. Hence human exposure to arsenic occurs via many routes. However, exposure is not well described in the literature, aside from exposure in occupational settings. The complex environmental chemistry of arsenic requires that the form, as well as the route, be considered in the evaluation of risk due to each exposure situation. This has not always been possible because the form(s) of arsenic involved in a specific route is not always known. Monitoring data for environmental media are most commonly reported for total arsenic concentrations rather than for specific chemical forms. This section addresses the various routes of arsenic exposure, as well as the form as specifically as possible. The magnitude, extent, and frequency of exposure associated with different routes are quantified to the degree the data permits.

Human beings have always been exposed to at least low levels of arsenic from natural background levels in various environmental media including water, soil, air and biota. Certain subpopulations may have evolved adaptations for living in local areas of high concentrations. There has been some speculation that arsenic at low levels may be an essential nutrient for mammals (NAS 1977); however this possibility has not been carefully investigated. Ideally, exposure of human subpopulations to industrial sources of arsenic should be assessed with an understanding of the population's previous exposure to background levels and consideration of the significance of the industrial input compared with arsenic levels already present.

5.2.2 Ingestion

5.2.2.1 Drinking Water

Exposure Levels

The maximum allowable contaminant level for arsenic in drinking water is 50 µg/l (U.S. EPA 1980a). Table 5-3 summarizes data on levels of arsenic found in drinking water in the U.S. In the Community Water Supply Survey for both surface and ground water supplies (U.S. DHEW 1970), only 0.2% of the samples contained arsenic at levels greater than 50 µg/l and only 0.4% were greater than 10 µg/l. The survey sampled 5% of the nation's water supply systems in 1970, of which 63% were ground water, 12% surface, 5% mixed, and the remaining 20% special systems. In other national surveys, few samples contain an arsenic level exceeding 10 µg/l (Greathouse and Craun 1978, U.S. EPA 1975). The mean level detected in 3834 samples was 2.37 µg/l (Greathouse and Craun 1978). Thus, it was assumed that most persons in the U.S. are exposed to concentrations of 2-2.5 µg/l with an exposure of approximately 4-5 µg/day. All of these surveys sampled for total arsenic.

A special survey of water supplies (presumably municipal) was conducted in six cities (26 samples) containing nonferrous smelters, a significant industrial source of arsenic (Kent 1976). Arsenic levels ranged from <5 µg/l to <30 µg/l. Therefore, these concentrations were comparable to those found in the national survey.

As can be seen, however, from Table 5-4 isolated cases of very high arsenic concentrations in ground water supplies have occurred. In some cases the sources are natural, as in Lassen County, California (Goldsmith et al. 1972), and Lane County, Oregon (Morton et al. 1976). These known levels are lower than those detected in other locations in the world. Only 5% of the Lane County levels exceeded 100 µg/l while 48% of the values in Taiwan did so (Mushak et al. 1980).

In general, there are relatively few data for ground water arsenic levels in the vicinity of industrial sources. In one case, the arsenic in ground water was attributed to buried arsenical pesticides (Feinglass 1973) (see Chapter 4.3). The arsenic found in the water supply of Ajo, Arizona may be due to contamination resulting from a nearby smelter. However, several other water supplies in Arizona towns with smelters contained levels of less than 10-20 µg/l (Baker et al. 1977). Recently,

TABLE 5-3. ARSENIC LEVELS DETECTED IN DRINKING WATER IN THE U.S.

Location	No. Samples	% Detected	Concentration (µg/1) Mean	Concentration (µg/1) Maximum	Source
U.S. (Community Water Supply Study--surface and well)	2595	0.2% > 50 µg/1 0.4% > 10 µg/1	--	100	U.S. DHEW (1970)
U.S. Drinking Water Survey (Residential Tap Water)	3834	66.8% < 0.1 µg/1	2.37	213.6	Greathouse and Craun (1978)
U.S. (Interstate Water Supply Study)	566	1% > 10 µg/1	--	60	U.S. EPA (1975)
Alaska--suburban community (well water)	59	--	224	2450	Public Health Service (1977)
Utah, Nevada, California (smaller communities)	--	--	--	10-330	Valentine (personal communication as cited in U.S. EPA 1979)
Perham, Minnesota (well water)	--	--	--	11,800-21,000	Feinglass (1973)
Lassen County, California (well water)	--	--	--	100-1400	Goldsmith *et al.* (1972)
Lane County, Oregon (well water)	558	8% > 50 µg/1	9.6	2150	Morton *et al.* (1976)
Ajo, Arizona (ground water at location of copper smelter)	--	--	--	70	Baker *et al.* (1977)
Wentatchee, Washington (well in area receiving lead arsenate treatment)	--	--	5.6		Fairhall (1941)
Bottled Mineral Water	--	--	20	190	Zoeteman and Brinkman (1976)

--: Not Available

TABLE 5-4. ARSENIC LEVELS DETECTED IN NON-POTABLE GROUND WATER IN THE U.S.[a]

Location	No. of Samples	Concentration (µg/l) Mean	Concentration (µg/l) Maximum	Reference
U.S. Major River Basins (1975-1979)	1922	2-22	500	STORET U.S. EPA (1980b)
Edgemont Well Study (1979): South Dakota and Wyoming[a]	13	27	67	STORET U.S. EPA (1980b)
Thermal Waters: Wyoming, Nevada, California, Alaska	--	range: 28-3800	3800	Fleischer (1963)

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[a] These data are included in preceeding U.S. data base.

in New England, a number of concentrations in drinking water wells have been reported in violation of the 50 μg/l drinking water criterion, with suspected arsenic-associated toxic effects in some consumers of the water supplies (Chow, C., personal communication February 17, 1981; Boston Globe 1981). The source of contamination is uncertain but suspected to be natural background levels in the soil or bedrock.

Ground water levels are also compiled in the USGS monitoring well data presented in STORET (see Chapter 4.0 Table 4-14). Table 5-4 presents the results. A total of 1922 observations (559 remarked) for the last 5 years in six river basins indicated mean major river basin levels ranging from 2 μg/l to 22 μg/l, with a maximum level of 500 μg/l in the Lake Erie basin. These data are not from potable water supplies, however, they are probably similar to arsenic levels in untreated drinking water from wells. The data points are too few and not well distributed enough nationally to indicate areas of high potential for human exposure. A local study (part of the major study) conducted in a minor river basin (Edgemont study) with a large number of high arsenic levels in all types of water reported all ground water levels less than 70 μg/l. The high levels in the basin were attributable to high surface water concentrations. It is unlikely that humans are exposed to the very high arsenic concentrations associated with areas of thermal activity shown in Table 5-4.

Table 5-3 suggests that levels up to 2000 μg/l may occur naturally in isolated water supplies, resulting in an exposure of 4000 μg/day. Exposures of this magnitude are probably limited to an extremely small sub-population. Whanger (1977) reports that "the natural occurrence of arsenic in ground water of Lane County is the only one in a well populated area of the North American Continent." However no extensive monitoring program that would support such a conclusion is known. Slightly more common exposures in known contaminated areas may be on the order of 200-400 μg/day.

The form of arsenic in water is usually soluble arsenates and arsenites. It is expected that most of the arsenic present in surface water sources of drinking water would be in the form of arsenate due to aeration during finishing. Clement and Faust (1973) reported that approximately 8% of the total arsenic in aerobic streams was in the trivalent form, while most of the arsenic in anaerobic waters was in this form. [The speciation diagram, Figure 4-3 (Chapter 4.0), supports this observation, showing almost 100% arsenite in soluble form under reduced conditions.] Anaerobic waters may contain arsine and methylated forms. Braman and Foreback (1973) report that methylarsonic acid and dimethylarsenic acid are found in surface waters. The form of arsenic in ground waters is not well studied, but Clement and Faust (1973) found that 25-50% of the arsenic in a limited number of ground water samples was in the trivalent form.

Potential Sources of Ground Water Contamination

Since little monitoring data for arsenic in ground water are available and considering the significance of land releases and arsenic's mobility under certain environmental conditions, it is relevant to review specific situations in which there is a potential for ground water contamination. The information in this subsection was discussed more thoroughly in Chapter 4 Pathway 2, but is reviewed here in an attempt to identify geographic areas in which ground water may be contaminated and human populations may be exposed to arsenic via ground water.

Table 5-5 summarizes releases by form, disposal practice and distribution for the major sources of arsenic releases to land. Maps of these distributions are compiled in Appendix D. Limited data are available on disposal practices, which are the key factor in determining whether or not arsenic will migrate out of the disposal site. Hence the various national regions listed are areas with potential ground water contamination. Further investigations are required to link actual incidences of contamination with sources. Information is also needed on typical solid waste disposal practices by industry.

Specifying the source of arsenic to land is only one factor, although an important one, in determining the potential for migration of the element to ground water. Once arsenic leaks, overflows or otherwise escapes from a disposal site, the surrounding soil and climatological characteristics become important determinants of the element's mobility. Table 5-5 lists environmental characteristics, both pedological and climatological, that are conducive to arsenic migration through soil. Appendix D includes a map of the national distribution of precipitation rates and soil types. The characteristics are meant to be illustrative only; migration will not occur wherever any one of these traits exists because it is dependent on a set of characteristics.

Comparison of Tables 5-5 and 5-6 suggests some areas in the U.S. where, based on loading rates and environmental characteristics, the potential for migration of arsenic into ground water appears to be high. These areas include the central eastern industrial states of Pennsylvania, southern New York, Ohio, Indiana, and other states where a large amount of arsenic is disposed of, a relatively high rate of precipitation exists, and the predominant soil type would appear to favor arsenic mobility. The southeastern states have high pesticide use rates and high precipitation rates. but a predominant soil type high in available ferrous and aluminum hydroxides which would tend to reduce arsenic mobility. The southwestern and Great Plains states have relatively low precipitation rates which would reduce mobility but soil low in available hydrous oxides. Washington, where disposal of arsenic production wastes are concentrated, has a high rate of precipitation and variable soil types, some of which are more conducive to migration than others.

TABLE 5-5. ENVIRONMENTAL RELEASES OF ARSENIC TO LAND; ESTIMATED ANNUAL VOLUME, CHEMICAL FORM AND GEOGRAPHIC DISTRIBUTION

Source	Quantity (kkg)	Predominant Form	Application Form	Distribution
Fossil Fuel Combustion	14,000	trioxide	ash ponds	National, 65% in industrialized northeast and central eastern U.S.
Pesticides				
• herbicide use	8,000	MSMA, DSMA, +3	directly to agricultural land	Approximately 85% of use in south central and southeastern U.S. (JRB 1980)
• cotton ginning	580	arsenious acid	landfill	Texas, Oklahoma
Copper Production	8,100	trioxide	sludge from tailing pond	Utah, Arizona, New Mexico, Montana, Nevada
Iron and Steel Production	5,700	trioxide	landfill or ponds	Primarily industrialized north and central eastern U.S. (PA, NY, OH)
Boron Production	2,200	trioxide	return to brine lakes	California
Manganese Production	1,400	unknown	slag	imported, waste disposal practices unknown
Arsenic Production	1,200	trioxide	slag piles or road construction material	Washington
Lead Production	1,100	trioxide	slag piles	Missouri, Colorado, Idaho, Utah, Other
Phosphorus Production	640	unknown	50% released in fertilizers applied to agricultural lands or land disposed	Tennessee, Florida, North Carolina, Western U.S. for wastes from production processes.
TOTAL	43,020			

TABLE 5-6. ENVIRONMENTAL CHARACTERISTICS FAVORABLE TO THE MIGRATION OF ARSENIC IN SOIL TO GROUND WATER

Characteristic	Potential distribution
Climatological	
High precipitation rate	Northeast, central East, Southeast and Northwest U.S.
Low rate of evapotranspiration	Northeast and Northwes.
Soil Characteristics	
Soils low in iron and aluminum hydrous oxides (low kaolinite clay content)	Soil orders: Inceptisols (S. NY, PA), Alfisols (IN, OH, WI), Mollisols (Northwestern and North Central Plains, Great Plains, Texas), and Aridosols (Southeast and Great Basin)
Reduced soils at pH 4-7	National distribution, intermittent occurrence
Permeable soils	Spodosols (N.E.) and Ultisols (S.E.)
High microbial activity	National distribution in temperate, moist areas, especially agricultural soils
High pH in aerobic soils	National distribution, more prevalent in western states

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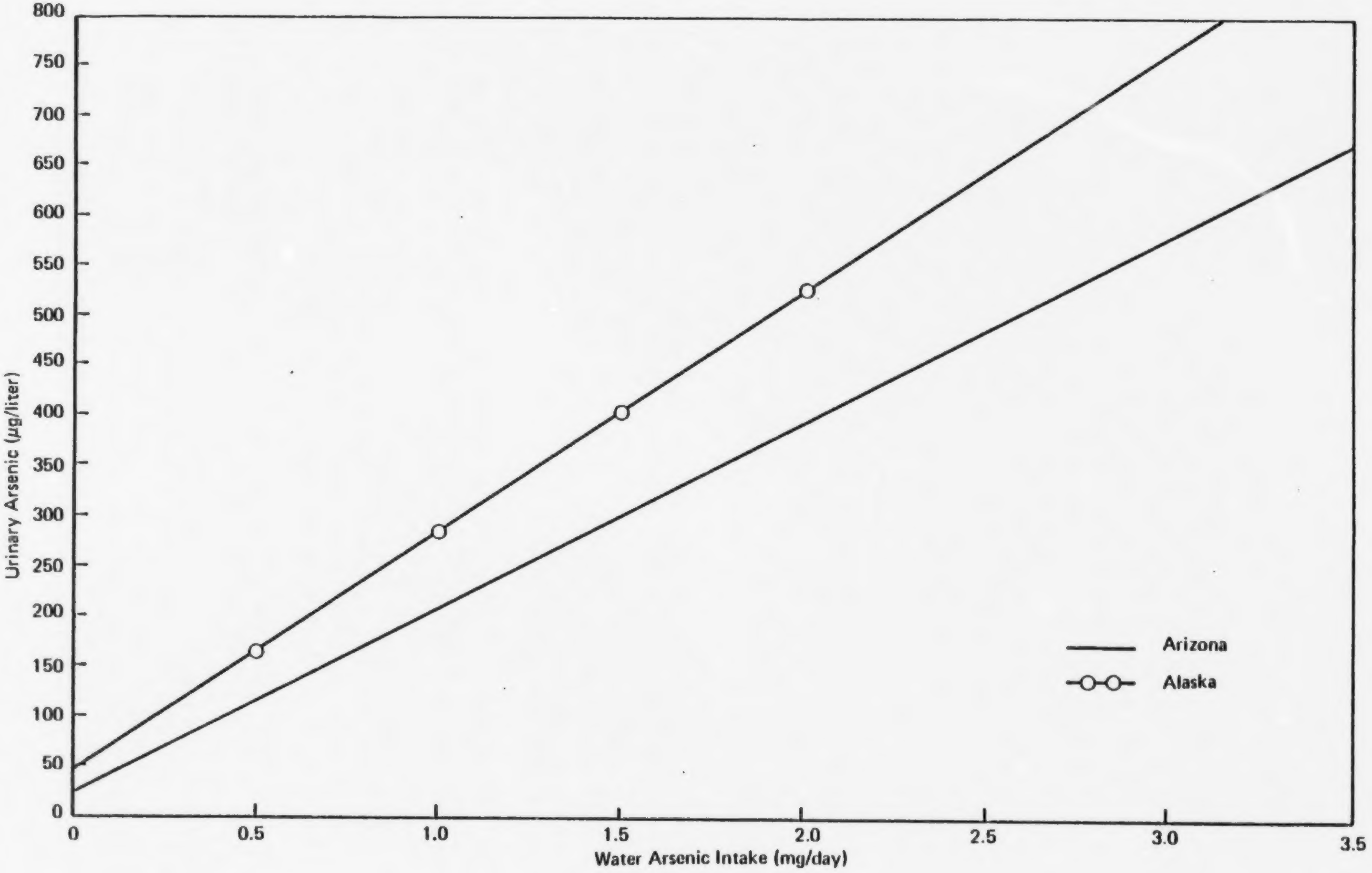
Source: Brady (1974); Arthur D. Little (1980); Eyre (1968).

This analysis is very cursory and has been made only for a first approximation of the potential for arsenic ground water contamination by region. Many other factors which are too variable to consider for a gross national-scale analysis--depth to water table, predominant arsenic form present due to site parameters, presence of fissures or cracks in soil through which mobility would be increased, and others--also have significant influences on the fate of land-disposed arsenic. Due to the large amount of arsenic in land wastes, the element's sometimes high mobility, and reported incidences of ground water contamination, a more detailed investigation of this subject is needed, with a focus on localized situations.

Relationship Between Exposure Levels and Urine Concentrations

A relationship between urinary levels of arsenic and rate of intake of arsenic in drinking water has been established by the U.S. EPA (Mushak *et al*. 1980). Figure 5-1 presents the regression curves for urinary levels versus daily consumption of arsenic in drinking water established from two studies in Arizona and Alaska. According to the graph, most of the U.S. population (exposed to 5 μg/day based on estimates in 5.2.2.1) would have arsenic levels of less than 100 μg/l in their urine. The subpopulation exposed to the maximum level of 4,000 μg/day would have urinary levels of from 650 μg/l to over 800 μg/l. If one compares arsenic urinary levels reported for people exposed to high arsenic levels--urinary levels which range from >300 μg/l to <700 μg/l (U.S. EPA 1980a)--(also see Chapter 5.2.4) with the levels on Figure 5-1, an intake level from drinking water can be estimated. This requires making the assumption that the levels present in the urine originate from ingestion of contaminated drinking water and not through inhalation of arsenic in air. This can be justified based on the small exposure levels associated with inhalation (at least two orders of magnitude below ingestion levels) for the general population, assuming that absorption efficiencies for the two routes do not differ by more than one order of magnitude. Making this assumption, the arsenic urinary level measured in the majority of the U.S. population (Gills *et al*. 1974), 22-25 μg/l, indicates a negligible intake of arsenic, confirming the conclusions based upon the limited monitoring data. Higher urinary levels resulting from exposure to contaminated drinking water in Chile (700 μg/l--Zoldivar and Guillier 1977) and in the vicinity of a smelter (539 μg/l--Pinto *et al*. 1976; 300 μg/l--Milhan and Strong 1974) indicate intakes on the order of 1 mg to 2.5 mg arsenic per day.

These extrapolations should be used with caution at this time and certainly require further investigation. The potential for uptake from sources other than drinking water or through other exposure routes exists and these factors could influence the apparent correlation between urinary levels and water concentrations.



Source: Mushak *et al.* (1980)

FIGURE 5–1 REGRESSION CURVES FOR URINARY ARSENIC VS TOTAL DAILY WATER ARSENIC INTAKE IN SUBJECTS IN ARIZONA AND ALASKA

5.2.2.2 Food

The sources of arsenic in food are numerous. The wide use of arsenic as a pesticide was an important source in the past; several cases of adverse effects from eating arsenic-treated fruit were reported (Cannon 1936). However, the pesticidal use of arsenicals has declined in recent years. The U.S. FDA allows 3.5 μg/l of arsenic in fruits and vegetables (NAS 1973). Concentrations of 100-500 μg/l arsenite have been measured in bottled wine (Crecelius 1977). A daily per capita consumption of 28 ml of wine has been estimated for the U.S. population over 21 years of age (Amerine and Singleton 1974). Assuming 100 μg/l in wine, this leads to an exposure of 0.004 μg/day for the average wine drinker. For the subpopulation ingesting 1 liter of wine daily with 500 μg arsenic/l, the exposure would be 500 μg/day. Crecelius (1977) found arsenite to be the predominant form in wine, at levels approximately five times greater than those of arsenate.

Also, finished moonshine was found to contain arsenic at levels as high as 415 μg/l (Gerhardt et al. 1980). An estimated 266 million liters of moonshine is produced each year (Ball and Sorenson 1969) so exposure may be more important than one would be inclined to think. Assuming ingestion of 28 ml/day, an exposure of 0.01 μg arsenic/day would result.

Arsenic is also present in meat and fish products. It is used as a growth stimulant for swine and poultry, accounting to some extent for levels found in these foods (Jellinek and Cornelieussan 1977). The FDA allows a maximum of 0.5 mg/kg in muscle tissue, 1 mg/kg in edible byproducts, and 0.5 mg/kg in eggs. Arsenical feed-additives are required to be removed from the livestock diet for the 5 days immediately preceeding slaughter.

Arsenic appears to accumulate in marine organisms, especially in bottom-feeding fish and crustaceans, which are exposed to both water and sediment levels of arsenic (see Chapter 4.0--bioaccumulation). A national monitoring survey indicated a mean of 2.6 mg/kg and a maximum of 700 mg/kg (Western Gulf) in freshwater and estuarine fish species (U.S. EPA 1980b). In marine waters, fish muscle tissue, livers, and whole body levels ranged typically from 2 mg/kg to 5 mg/kg and crustaceans from 4 mg/kg to 5 mg/kg (wet weight--Hall et al. 1978).

The FDA (1977) has estimated that in 1974 the total dietary intake of arsenic in food was 21 μg/day (as As_2O_3). In 1973, it was estimated at 10 μg/day, although the authors did not feel that this represented a significant difference. This average intake has decreased from an estimated 63 μg/day, for the years 1965-1970, mainly due to a decline in residues in fruits and vegetables. The decline presumably resulted from a reduction in use of arsenical pesticides during this period. Most of the arsenic intake in 1974 (79%) was attributed to meat, poultry, and fish. Seafood, especially bottom-feeding fish and crustaceans, usually contains levels of 1-10 mg/kg arsenic, although others report a range of 1-100 mg/kg (Munro 1976; Lunde 1977). Schroeder and Balassa (1966) estimated that a diet without seafood could contribute 40 μg/day, while with seafood would result in an exposure of 190 μg/day. This appears to be in the range of FDA estimates for that time.

The arsenic content of various specific food items is discussed in various other sources and in Section 4.4. Thus, these results will not be repeated here.

Jellinek and Corneliussen (1977) examined various foods within groups. Their results are shown in Table 5-7. It can be seen that levels in most foods are in the range of 0.01-0.04 mg/kg, with the exception of finfish. A worst case exposure might be estimated by assuming a maximum level of 100 mg/kg of arsenic in fish, and assuming a consumption of 100 g fish/day. The resultant exposure would be 10,000 μg/day. This estimate probably represents a somewhat unrealistic upper bound estimate, since fish tissue levels are usually less than 10 mg/kg. In addition, only a very small subpopulation consumes 100 g/day of fish.

There is much uncertainty regarding the form of arsenic in food. Crecelius (1977) investigated the form of arsenic in wine. The trivalent form was found at levels of 1-420 μg/l; the pentavalent form was found at levels of 1-110 μg/l. Total arsenic was found at levels of 1-530 μg/l, and about 50% of the samples contained more than 50 μg/l, thus suggesting wine as a signficant source of human exposure, especially to the trivalent form. Both methylarsenic acid and dimethylarsenic acid were undetected at 1 μg/l. The author speculated that As^{+5} was taken up by plants, and then converted to As^{+3} during fermentation. The pentavalent form is expected to be the soluble form of arsenic most commonly available for plant uptake from soil (see Chapter 4.0).

Little is known regarding the form of arsenic in other foods, with the exception of seafood. Lunde (1977) has attempted to identify the compounds found in seafood and has concluded that they are in the form of several lipid-soluble organic compounds and one water-soluble organic compound. Uthe and Reinke (1975) demonstrated that transformation between chemical forms of arsenic may take place between the time of death for the fish and consumption by humans. Tissue in dead fish, shellfish and lobster supported chemical reduction of arsenate to arsenite to the point that the trivalent form was the predominant one. The time required for such extensive transformation was not given. Typical cold storage practices which would slow down any reaction and consumption of usually fresh fish, make it unlikely that arsenite would be prevalent in much of the fish and shellfish consumed by the U.S. population.

Due to the propensity for arsenic to be transformed into the volatile trioxide form when subjected to heat, it is important to know for human exposure whether signifcant losses of arsenic may occur during

TABLE 5-7. ARSENIC LEVELS IN FOODS

Food Group	As_2O_3 Levels (mg/kg)[a] Mean	Maximum
Meats, Eggs and Milk	0.01-0.03	0.5 - chicken
Vegetables and Fruits	0.01-0.03	0.3 - potato products
Cereal, Nuts and Sugar Products	0.01-0.04	0.4 - rice
Finfish and Shellfish	0.07-1.47	19.1 - finfish

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[a] Note: Arsenic levels are reported as concentrations of As_2O_3.

Source: Jellinek and Corneliussen (1977).

the cooking of contaminated fish or other foods. However, Anand (1978) found no loss in arsenic concentration following the frying of fish for 7 minutes. There was no information on the effect of longer heating times or higher temperature on arsenic losses.

5.2.2.3 Soil

Exposure through ingestion of contaminated soil may occur in certain presumably small, subpopulations. Children are more likely to be exposed than adults. One major source of arsenic in soil is pesticidal applications. In 1967, the USDA put restrictions on use of certain arsenicals in domestic pesticide products (NAS 1977). Products containing more than 2.0% sodium arsenite and 1.5% arsenic trioxide are no longer registered for home use. There is still a potential for exposure to arsenic in soil treated with the organic arsenicals: DMSA, MSMA and cacodylic acid. Approximately 8% of the MSMA pesticides are used domestically (JRB 1980). Most of the use likely to result in non-occupational exposure is expected to be by institutions in lawn treatment.

Assuming retention in the top 1 cm of soil following turf treatment at the prescribed rate (see Chapter 4.0, Pathway 5), the upper level concentration of arsenic in soil to which humans would be directly exposed is 2100 mg/kg. A child ingesting 10 mg of soil per day (Lepow et al. 1975) would ingest approximately 0.02 μg of arsenic, presumably in one episode. The likelihood of such an exposure immediately following turf treatment is expected to be quite low. Ingestion of soil with natural levels of arsenic or much lower treatment levels would result in negligible exposure. Grass concentrations would be lower than soil levels (see Chapter 6.0), probably no greater than 10 mg/kg, so exposure through accidental ingestion is expected to be minimal.

5.2.3 Inhalation

The average daily concentration of atmospheric arsenic at 267 urban locations in the United States was 0.003 $\mu g/m^3$ in 1974 (Suta 1980). For an assumed respiratory flow of 20 m^3/day, an exposure of 0.06 μg/day would result. Some urban areas with nonferrous smelters within a 50-mile radius have higher atmospheric levels, as much as 0.08 $\mu g/m^3$ (in Tacoma, Washington) which would result in an exposure of 1.6 μg/day (Suta 1980). The average annual concentration for eight rural areas was 0.0004 $\mu g/m^3$ (Suta 1980), which would result in a comparatively negligible exposure. The form of arsenic, especially in industrial areas, is generally particulate, as inorganic oxide or arsenite (Suta 1980).

Concentrations of arsenic are also elevated in the immediate vicinity of production facilities for arsenic compounds, copper smelters, and cotton gins. Milham and Strong (1974) reported that arsenic content of dust was as high as 1300 mg/kg within 0.4 miles of the Tacoma, Washington smelter, while it declined to 70 mg/kg at 2.0-2.4 miles from the source.

Suta (1980) conducted an exposure analysis of the nonoccupational subpopulations exposed to airborne concentrations of arsenic from various sources both estimating exposure levels and the size of each subpopulation exposed. Table 5-8 summarizes his conclusions. Suta estimated that upper limit atmospheric concentrations in the vicinity of most emission sources were approximately 0.3 μg/m^3 at locations where people would be exposed (e.g., ground-level, outside the plant). For a 24-hour period of exposure, this level would result in an exposure of 6 μg/day. Copper smelters and cotton gins may expose subpopulations to higher levels, as much as 20 μg arsenic/day. The chemical form of exposure is presumably arsenic trioxide, commonly associated with thermal processes. An exception is the arsenic form generated by cotton ginning, presumably arsenious acid.

Arsenic is also present in cigarettes (Lee and Murphy 1969), and smoking may result in low levels of exposure. In the past, the use of arsenic-containing pesticides on tobacco resulted in levels of up to 42 μg arsenic per cigarette (Lisella et al. 1972). Since 1952, however, arsenicals are no longer used on tobacco, and the arsenic content in one cigarette is now less than 12 μg. Lisella et al. estimated that 15% of the arsenic present would be volatilized (presumably as trioxide). If 50 cigarettes are consumed per day and all volatilized arsenic is inhaled, the daily exposure level would equal 90 μg of arsenic.

Exposure via inhalation of arsenical pesticides or of dust or vapors from arsenic-treated wood may occur in small non-occupational subpopulations. The RPAR position document on inorganic arsenical pesticides estimated an exposure level of 270 μg of arsenic per 8-hr day from handling, sawing and fabricating with arsenic-treated wood products (U.S. EPA 1978). Exposure from living in a home built with treated wood was estimated at 0.7 ng to 2 μg per day from inhalation of vapors.

5.2.4 Dermal Contact

Dermal contact with arsenic would be expected to be minimal, since water concentrations are generally low. However, Angino et al. (1970) pointed out that dermal exposure may result from the use of detergent containing arsenic. These authors calculated that the highest concentration of arsenic would be found in a 1-gal presoak solution, which might contain 20-250 mg/l of arsenic. Arsenic has been found at up to 70-80 mg/kg in phosphate-containing detergents (Pattison 1970). The form present is thought to be pentavalent. At typical washwater concentrations, arsenic at the highest level was estimated to be present at approximately 0.15 mg/l. Assuming a dermal absorption rate of 23.2 mg/hr (for arsenate measured in a 15 g/l solution; see Section 5.1.2.1), high enough arsenic concentrations for optimal uptake, and an exposure period of 10 minutes, the resulting uptake of arsenic would be approximately 4 mg. This number is most likely an overestimation of exposure due to the 5 order of magnitude difference between arsenic concentrations in washwater and the concentration in the solution in which the absorption rate was measured. Also assuming a washing machine or tub capacity of 60 l, the total amount of arsenic available for absorption would be approximately 9 mg. The estimated exposure level suggests that 45% of this amount is absorbed in the 10 minute period of exposure which is a probable overestimate. Therefore, there is a high degree of uncertainty associated with this estimate.

TABLE 5-8. SUMMARY OF MAGNITUDE OF HUMAN POPULATIONS EXPOSED VIA INHALATION OF ARSENIC BY SELECTED EMISSION SOURCES

Average Annual Concentration[a] ($\mu g/m^3$)	No. Persons Exposed by Emission Source					
	Copper Smelters[b]	Lead Smelters[c]	Zinc Smelters[d]	Cotton Gins[e]	Pesticide Manufacturer[f]	Glass Manufacturer[g]
3.0-5.9				2		
1.0-2.9				30		
0.60-0.99	24,420			70		
0.30-0.59	19,380			230		
0.10-0.29	137,450	800	9,000	660		1,440
0.060-0.099	119,860	2,600	18,000	990		10,140
0.030-0.059	286,560	5,100	101,000	1,900		75,180
0.010-0.029	313,380	38,000	170,000	6,600		212,040
0.005-0.009	8,810	46,000	110,000	18,000	380	363,870
0.003-0.004	12,960	67,000	13,500	44,000	1,100	583,360

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[a] Average omnidirectional concentrations. With the exception of cotton gin exposures, 24-hr worst-case exposures can be estimated by multiplying the annual averages by 12.5. The 24-hr worst-case exposures for cotton gins may be obtained by multiplying the concentrations by 81.

[b] Based on EPA's estimate of emissions.

[c] Based on an emission of 0.8 lb of arsenic per each ton of lead produced. Fugitive emissions are estimated to be 10% of stack emissions.

[d] Based on an emission of 1.3 lb of arsenic per ton of zinc produced by pyrometallurgical smelters and no stack emissions at electrolytic smelters. Fugitive emissions assumed to be 10% of stack emissions.

[e] Annual average exposure, assuming that ginning exposures occur during 15% of the year and that there are no exposures during the remainder of the year.

[f] Assumes that 111 large plant pesticide emissions are well controlled.

[g] Assumes that 15% of pressed and blown glass is manufactured with arsenic, that only certain manufacturers use arsenic in all of their pressed and blown glass production, and that the size distribution of manufacturers who use arsenic is proportionate to the size output. It is assumed that 90% of the manufacturers are well-controlled and that 10% are poorly-controlled.

Source: Suta (1980).

Additional exposure may occur from the handling of arsenic-treated wood (Fowler et al. 1979) or of grass clippings from arsenic-treated lawns. Exposure levels associated with these activities are usually presumably low because the arsenic is not available in a solution in which it would be most likely to be absorbed dermally. The RPAR position document on inorganic arsenical pesticides reported an estimated exposure level of 240 μg in an 8-hr day associated with handling, sawing and fabrication of arsenic-treated wood products (U.S. EPA 1978). This estimate assumes that sweating of skin during labor would solubilize the arsenic absorbed by the wood.

5.2.5 Exposure Incidences as Indicated by Human Monitoring Data

Elevated levels of arsenic appear in human hair and nail following long-term exposure to the element; however relating these accumulated levels to effects levels has not been successful so far. Metabolic studies indicated little tissue accumulation for most forms of arsenic. The effect of arsenic on the body by the time it is excreted in dead hair and nail matter is unknown. Relating exposure to urinary levels from ingestion of contaminated drinking water was described in 5.2.2.

Table 5-9 presents reported data on arsenic levels in human biological media: hair and urine. The general population and selected subpopulations exposed to specific sources are represented. The majority of the exposure incidents resulting in bioaccumulation were related to inhalation of airborne arsenic. Other exposure incidents that resulted in adverse effects were described in 5.1.4.

5.2.6. Forms of Arsenic Associated with Exposure Pathways

A wide variety of chemical forms of arsenic are found in the environmental media to which humans are exposed. Since speciation is a significant factor in arsenic toxicity (see 5.1), it is ideal to identify the particular form(s) to which humans are exposed to estimate the risk associated with each pathway. This report has focused primarily on those forms thought to be most environmentally significant in terms of toxicity and abundance. These include arsenate, arsenite, MSMA, DSMA, cacodylic acid, unidentified organically bound arsenic in biota, and arsine. In this section, mention was made of the particular form(s) of arsenic known or suspected to be associated with each exposure pathway, whenever possible. Table 5-10 summarizes, by form of arsenic, the exposure pathways in which each form may be present. Both quantitative and non-quantitative observations are included whether or not exposure levels could be calculated based on data. Arsenate and arsenite are common forms in water but their significance in foods is generally not known. The organic arsenicals are present in treated crops and some water samples. Organic-bound arsenic, usually bound to sulfhydryl groups, is found in plant and animal tissue. Arsine is rarely found and only under very anaerobic conditions.

TABLE 5-9. ARSENIC IN HUMAN BIOLOGICAL MEDIA

Tissue	Number of Cases	Tissue Arsenic Levels		Source	Comments	Reference
		Range	Mean			
HAIR	a) 44 b) 69 c) 14 d) 5 e) 25	a) >1 ppm b) ≤1 ppm c) >2 ppm d) >1 ppm ≤2 ppm e) ≤1 ppm	NG*	92 people using wells with high As; 21 people As less than 0.05 ppm	a) 17/44 As poisoning b) 10/69 As poisoning c) 7/14 Electromyograph abnormal d) 3/5 " " e) 0/25 " "	Hindmarsh et al. (1977)
HAIR	a) 10 b) 93 c) 26	NG	a) 0.32 mg/100g b) 0.61 mg/100g c) 0.08 mg/100g	Geological contamination of well water	a) arsenic area, normal skin b) arsenic area, abnormal pigment c) nonarsenic area, normal skin	Borgono et al. (1977)
HAIR	13	37-1,680 µg/g	NG	Well water contaminated with insecticide	Arsenic in water 11,800-21,000 mg/liter	Feinglass (1973)
HAIR	220	a) 2.8-13 ppm b) 0-1.5 ppm c) 0.4-2.8 ppm	NG	Soy sauce contaminated with arsenic (perhaps calcium arsenate)	a) near the root b) at the tip c) control	Mizuta et al. (1956)
HAIR	a) 23 b) 23 c) 44	a) 0.6-8.1 µg/g b) 0-0.9 µg/g c) 0-1.1 µg/g	a) 3.3 µg/g b) 0.3 µg/g c) 0.2 µg/g	Air pollution from coal-fired power plant	a) heavily polluted area b) 36 km from source c) controls	Bencko and Symon (1977)
HAIR	a) 872 b) 160	NG	a) 2.6 µg/g b) 0.9 µg/g	Stack emissions from non-ferrous smelters	a) smelter area b) control	Baker et al. (1977)

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*NG: data not given.

continued...

TABLE 5-9. ARSENIC IN HUMAN BIOLOGICAL MEDIA
(Continued)

Tissue	Number of Cases	Tissue Arsenic Levels: Range	Tissue Arsenic Levels: Mean	Source	Comments	Reference
HAIR	a) 39 b) 74 c) 94 d) 48 e) 48 f) 123	a) NG b) NG c) NG d) 20-1550 μg/100g e) 27-6014 μg/100g f) 34.8-363 μg/100g	a) 668.9 ± 500.9 μg/100g b) 334 ± 401 μg/100g c) 794.4 ± 1244.7 μg/100g d) 287.2 μg/100g e) 730.9 μg/100g f) NG	Air pollution	a) Zn sulfate process b) Cd-Zn refinery c) Cd-Zn refinery d) Cd-Zn refinery e) Cd-Zn refinery f) residents of 5 urban areas	Gabor and Coldea (1977)
HAIR	a) 13 b) 7	a) 19-99 b) 0-5 ppm	a) 61 ppm b) 1.3 ppm	Stack emission from copper smelter	a) children 300 yd from source b) children 8 miles from source	Milham and Strong (1974)
HAIR	61	NG	a) 1.7 μg/g b) 1.9 μg/g c) 49.5 μg/g d) 0.3 μg/g	Arsenic mining	a) roasters/smelters b) ore dressers c) miners d) clerical	Ishinishi, *et al.* (1977)
HAIR	a) 21 b) 20	a) 0.3-6.1 ppm b) 0.08-0.18 ppm	a) 2.1 ppm b) 0.12 ppm	Emissions from zinc and copper mining	a) children near mine b) urban controls	Corridan (1974)
HAIR	310	0.08-15.15 mg/100 g	1 mg/100 g	Geological contamination of drinking water	Normal value reported 0.1 mg/100 g	Zaldivar and Guillier (1977)
HAIR	a) 1 b) 1	NG	77 ppm 5 ppm	Contamination of well-water by waste from arsenic sulfide plant	a) no skin symptoms b) advanced skin symptoms	Terada (1960) Tsuchiya (1977)
URINE	164	0.001-0.7 mg/liter	0.09 mg/liter	Geological contamination of drinking water	Maximum normal value reported 0.1 mg/liter	Zaldivar and Guillier (1977)

continued...

TABLE 5-9. ARSENIC IN HUMAN BIOLOGICAL MEDIA (Continued)

Tissue	Number of Cases	Tissue Arsenic Levels		Source	Comments	Reference
		Range	Mean			
URINE	1	a) 0.5-11 ppb b) 0.5-11 ppb c) 8-39 ppb d) 3-17 ppb	NG	Wine contaminated with arsenic	a) As^{+3} b) As^{+5} c) DMAA d) MAA	Crecelius (1975)
URINE	a) 25 b) 20 c) 44	a) 0.001-0.105 μg/g b) 0-0.036 μg/g c) 0.001-0.044 μg/g	a) 0.025 μg/g b) 0.008 μg/g c) 0.011 μg/g	Air pollution from coal-fired power plant	a) heavily polluted area b) 36 Km from source c) controls	Bencko and Sy (1977)
URINE	a) 712 b) 193	NG	a) 18.7 μg/l b) 5.8 μg/l	Stack emissions from nonferrous smelters	a) smelter area b) control	Baker et al. (1977)
URINE	a) 49 b) 78	NG	a) 0.021 ± 0.017 mg/ b) 0.023 ± 0.032 mg/	Air pollution	a) Zn sulfate process b) Cd-An refinery	Gabor and Coldea (1977)
URINE	a) 19 b) 16 c) 7 d) 6	a) 0.01-0.3 ppm b) 0.01-0.2 ppm c) NG d) NG	a) 0.097 ppm b) 0.034 ppm c) 0.30 ppm d) 0.02 ppm	Stack emission from copper smelter	a) children 300 yd from source b) children 8 miles from source c) 0-0.4 miles d) 2-2.4 miles	Milham and Strong (1974)
URINE	a) 15 b) 19	a) 8-49 μg/day b) 11.5-75.5 μg/day	a) 20.9 μg/day b) 39.1 μg/day	Urban areas	a) Seattle, 1965 b) Denver, 1972	Mathies (1974)
URINE	a) 41 b) 30 c) 23 d) 30	NG	a) 1.3 μg/l b) 2.2 μg/l c) 4.8 μg/l d) 8.6 μg/l (geometric means for As^{+3})	Copper smelter workers	a) controls b) low exposure c) medium exposure d) high exposure	Smith et al. (1977)

continued....

TABLE 5-9. ARSENIC IN HUMAN BIOLOGICAL MEDIA
(Continued)

Tissue	Number of Cases	Tissue Arsenic Levels: Range	Tissue Arsenic Levels: Mean	Source	Comments	Reference
URINE	24	33-539 µg/l	174 µg/l	Copper smelter	Mean airborne As 53 $\mu g/m^3$; increases associated with seafood consumption	Pinto et al. (1976)
URINE	a) 5 b) 5 c) 5 d) 5 e) 5 f) 5 g) 5	a) 13-77 µg/24 hr b) 104-256 µg/24 hr c) 81-519 µg/24 hr d) 8-212 µg/24 hr e) 24-201 µg/24 hr f) 11-123 µg/24 hr g) 4-89 µg/24 hr	a) 38 µg/24 hr b) 172 µg/24 hr c) 204 µg/24 hr d) 68 µg/24 hr e) 73 µg/24 hr f) 42 µg/24 hr g) 26 µg/24 hr	5 occupationally exposed Forest Service workers	a) 1st week exposure b) 4th week exposure c) 6th week exposure d) 8th week exposure e) 10th week exposure f) 2nd week control g) 6th week control	Wagner et al. (1974)
URINE	NG	0.022-0.025 mg/l	0.023 mg/l	NG	"Normal" freeze-dried	Gills et al. (1974)

TABLE 5-10. CHEMICAL FORMS OF ARSENIC ASSOCIATED WITH DIFFERENT EXPOSURE MEDIA

	Arsenate	Arsenite	MSMA, DSMA, Cacodylic Acid	Organic-bound Arsenic	Arsine
Surface Drinking Water	Predominant form	Negligible, epecially under aerated conditions	May be present at low levels	NP[a]	NP
Ground Drinking Water	May be major form in aerated waters	May be predominant form under reducing conditions	May be present at low levels	NP	May be present in very reduced conditions
Ambient Water	Usual predominant form	Usually present, sometimes significant	Usually present, sometimes significant	Unknown	NP
Food (general)	ALL FORMS EXPECTED TO BE PRESENT				
Fish	Unknown	Present in some fish tissues	Predominant soluble form	Predominant form	NP
Meat	Unknown	NP	May be present at trace levels in animals fed feed additives	Predominant form	NP
Crops	Unknown	Unknown	Predominant form	Predominant form	NP
Wine	Present at low levels	Predominant form	Unknown	NP	Unknown
Atmosphere	NP	Predominant form (As_2O_3)	Unknown	NP	NP
Cigarette Smoke	NP	Predominant form (As_2O_3)	Unknown	NP	NP
Arsenic-treated Wood	Predominant form (chromated copper arsenate and fluor-chrome arsenate phenol)	NP	NP	Unknown	NP

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[a] NP means not thought to be present.

5.2.7 Summary

The typical and maximum arsenic levels associated with various human exposure pathways are summarized in Table 5-11. Data are grouped by each main exposure route: ingestion, inhalation and dermal absorption. The size of the subpopulations expected to be exposed to each pathway is estimated as best as possible. Also included are the assumptions on which the estimated exposure levels are based and chemical form of arsenic.

Most typical exposure levels equaled 20 µg arsenic per day or less for all routes. Levels in excess of 100 µg/day were estimated for ingestion of drinking water and food, working with treated wood and use of contaminated detergents. Exposure to 4000 µg/day may occur with ingestion of ground water with arsenic levels equivalent to levels in areas with high background concentrations or in the vicinity of disposal sites. Subpopulations consuming large amounts of contaminated fish may be exposed to very high levels of arsenic, as much as 10,000 µg/day. Exposure through inhalation and dermal contact during construction with treated wood had associated levels of 260 µg and 240 µg per day. Dermal exposure to highly contaminated detergent in solution had an estimated uptake of 4000 µg, however, there is some uncertainty regarding the applicability of the arsenic absorption rate at the arsenic concentrations present in washwater. All exposures to atmospheric arsenic had low exposure levels, less than 20 µg/day, even in the vicinity of sources. Approximately 90 µg/day was the exposure estimated for a heavy cigarette smoker.

TABLE 5-11. ESTIMATED LEVELS OF HUMAN EXPOSURE TO ARSENIC

Route	Probable Form	Exposure (ug/day)		Subpopulation Exposed	Assumptions
		Typical	Maximum		
Ingestion - Drinking Water					
Surface sources	Arsenate	5	200	General population which consumes surface water supplies: 117×10^6 (Temple, Barker and Sloan 1977)	Typical: Most levels (99.6% of D.W. survey) in U.S. <10 ug/l; mean = 2.5 ug/l; consumption of 2 l/day. Maximum: Maximum level in drinking water supply 100 ug/l; consumption of 2 l/day.
Ground water sources	Arsenite from 25%-100%; MSMA, DSMA, some arsenate in less reduced water. Arsine has been detected under very reduced conditions	5	4000	General population which consumes ground water supplies: 75×10^6 (Temple, Barker and Sloan 1977).	Typical: Small sample of average groundwater levels at 10 ug/l or less; consumption of 2 l/day. There are many incidences of higher ground water levels (See text) Maximum: Maximum levels of 2000 ug/l in naturally contaminated supplies, consumption of 2 l/day.
Food -- total diet	All forms; large part may be organically bound arsenic	21	190	General population: 221×10^6 (USDC 1980).	Typical: FDA Total Diet study estimate. Maximum: Total diet with seafood (Shroeder and Balassa 1966).
High fish consumption	MSMA, possibly arsenite and others	1000	10,000	Very small.	Typical: Fish or shellfish containing 10 mg/kg; consumption of 100 g fish/ Maximum: Fish or shellfish containing 100 mg/kg As; consumption of 100 g fish/day
Moonshine consumption	Unknown	0.01	415	Very limited subpopulation	Typical: Moonshine containing 415 ug/l; consumption of 28 ml/day. Maximum: Moonshine containing a maximum of 415 ug/l; consumption of 1 l/day
Wine consumption	Arsenite predominately and arsenate	0.004	500	General population	Typical: Wine containing 100 ug/l; consumption of 28 ml/day. Maximum: Wine containing maximum levels of 500 ug/l; consumption of 1 l/day.
Soil ingestion	Arsenate or organic arsenicals	0.02	20	1/3-1/2 of children up to age 3 (Mahaffey, 1978).	Typical: Soil containing 2100 mg/kg; consumption of 10 mg soil/day. Maximum: Soil containing 2100 mg/kg; consumption of 10 mg soil/day.
Inhalation					
General atmosphere	Arsenic trioxide	0.06	0.6	General population: 221×10^6 (USDC 1980).	Typical: Average ambient concentration of 0.003 ug/m^3; respiratory flow of 20 m^3/day. Maximum: Typical urban concentration in cities (containing smelters) of 0.03 ug/m^3.
Vicinity of industrial sources	Arsenic trioxide	6	20	See Suta (1980) estimates by industrial source	Typical: Atmospheric concentration of 0.3 ug/m^3. Maximum: Atmospheric concentration of 6 ug/m^3. Respiratory flow of 20 m^3/day. May be higher exposure levels at some sites.
Living in house built of arsenic-treated wood	Unknown	< 0.001 to 2	--	Very small subpopulation	Range: See U.S. EPA (1978) RPAR document on arsenical pesticides
Cigarette smoking	Arsenic trioxide	--	90	Smokers: 54.1×10^6 (Surgeon General 1979).	Arsenic concentration of 12 ug/cigarette 15% volatilized; consumption of 50 cigarettes/day
Handling, sawing, building with arsenic-treated wood	Unknown; form is chromated copper arsenate and fluor chrome arsenate phenol (see 3.3.2)	--	260[1]	Very small subpopulation	See U.S. EPA (1978) RPAR document on arsenical pesticides.
Dermal Absorption					
Handling, sawing, building with arsenic-treated wood		--	240[1]	Very small subpopulation	See U.S.EPA (1978) RPAR document on arsenical pesticides. Assumes uptake of 0.529 mg arsenic per square foot of wood, 8 hour exposure, damp palms
Use of arsenic-contaminated detergent.		--	4000	Very small subpopulation	Assuming a dermal absorption rate of 23mg hr (based on measurement in solution of 15 g arsenic/l) and exposure of hands for 10 minutes. There is a high degree of uncertainty associated with this estimate (see Section 5.2.4).

*Includes absorption efficiency across skin.

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6.0 EFFECTS AND EXPOSURE--NONHUMAN BIOTA

6.1 Effects on Nonhuman Biota

6.1.1 Introduction

This section provides information about the levels of arsenic exposure at which the normal behavior and metabolic processes of aquatic organisms, terrestrial plants, and microorganisms are disrupted, as indicated by laboratory and field studies. There is a fairly extensive data base available for some of the chemical forms of arsenic; however, virtually nothing is known about the toxicity of other forms. In addition, the chemistry of arsenic in aquatic systems is complex (as described in Chapter 4.0) so that laboratory studies are often too simple to represent actual environmental conditions. Arsenic is usually presented as a salt in bioassays, dissolved in distilled or filtered water so that the majority of the total concentration is available for biological uptake. In natural conditions, complexing and precipitation with ferrous and aluminum hydroxides and adsorption onto clay will remove some of the available arsenic from solution. Therefore, effects levels measured in the laboratory may be lower than those in the environment.

In general, the pentavalent forms of arsenic are expected to be most prevalent in the aerobic, neutral waters that make up the habitat for most aquatic species. Sodium arsenite (+3) is used as an aquatic herbicide and so may expose aquatic biota immediately after application. Transformation to the arsenate (+5) form would occur soon after release in aerobic systems. Biota living in slightly reduced waters, such as in eutrophic lakes or ponds or in the surface sediment layer, may be exposed to the trivalent form. Deeper in the sediment, burrowing organisms are theoretically exposed to arsine (-3); however, no information on concentrations of this form was available.

The toxicology data for fresh and saltwater biota are fairly extensive, representing a number of fish and invertebrate species. Most bioassays were conducted with the inorganic trivalent form (arsenite or arsenic trioxide). A few studies were available on the pentavalent (+5) form (arsenate), organic arsenicals, and arsenic trisulfide. No data were available on the toxicity of arsine forms.

Most of the information presented in this section is based on the U.S. EPA (1980a) Ambient Water Quality Criteria Document for arsenic. Other reviews concerning the aquatic toxicity of arsenic can be found in the U.S. EPA (1978) position document on wood preservative pesticides, in Caldwell et al. (1976) and Spehar et al. (1980).

6.1.2 Freshwater Biota

The Criteria and Standards Division of U.S. EPA has set a freshwater criterion for arsenic of 0.44 mg/l for protection of aquatic life (U.S. EPA 1980a). The criterion applies to total recoverable trivalent inorganic arsenic. Short-term freshwater effects were observed at levels as low as 0.04 mg/l. No criterion was set for protection of saltwater life; however toxic effects were reported at levels of 0.51 mg/l.

The mechanism of active toxicity differs among arsenic compounds. The primary mechanism for the trivalent form is believed to be inactivation of enzymes due to reaction with the sulfhydryl groups of proteins. The arsenate form is not as reactive with sulfhydryl groups but has been found to uncouple oxidative phosphorylation.

Seven fish species have been tested with sodium arsenite. The range of acute toxicity values was narrow for these species: the LC_{50} (concentration lethal to 50% of test organisms) ranging from 13.4 mg/l to 41.8 mg/l. The rainbow trout was the most sensitive fish tested (LC_{50} of 13.3 mg/l) and bluegill the most resistant (LC_{50} of 41.8 mg/l). Sodium arsenate was tested on rainbow trout and a LC_{50} of 10.8 mg/l was determined. Sodium arsenate was toxic to green sunfish (Lepomis cyanellus) at a concentration of 150.0 mg/l; otherwise the range of toxicity values to fish from arsenite and arsenate was similar. Toxicity levels for other freshwater species (crayfish, channel catfish, and smallmouth bass) exposed to monosodium methanearsonate (an aquatic herbicide) were very high, a finding indicating that organic arsenic may be much less toxic than either inorganic arsenite or arsenate (U.S. EPA 1980a). Selected data for freshwater species are summarized in Table 6-1. The reader is referred to U.S. EPA (1980a) for further data.

Freshwater invertebrates tested for acute toxicity of arsenic include four cladocera and a scud and one aquatic insect. The cladocera and scud (LC_{50} range 0.8-5.3 mg/l) were approximately four times as sensitive to sodium arsenite as the stonefly (22.0 mg/l) (Table 6-2). Other data on the effects of several arsenicals to a variety of invertebrates are summarized in U.S. EPA (1980a). In general, reduced populations of freshwater zooplankton and insects are reported for arsenic concentrations in the range of 2.2-11.1 mg/l.

Data on chronic effects were available only for Daphnia magna. A life-cycle test was conducted with both sodium arsenite and sodium arsenate; the chronic values for both arsenicals were 0.9 mg/l indicating, once again the similar toxicity of the two compounds for aquatic biota (U.S. EPA 1980a).

Sodium arsenite, which is often used as an aquatic herbicide, was tested with the eggs and fry of the freshwater Muskellunge (Esox masiquinongy). Fifty percent of newly-hatched fry in 5.0 mg/l were dead on day 9; 47% mortality on day 12 occurred at a concentration of of 1.0 mg/l; and a concentration of 0.05 mg/l yielded a 46% mortality

TABLE 6-1. ACUTE TOXICITY OF ARSENIC FOR FRESHWATER FISH

Organism	Test Compound	Bioassay Method[a]	Hardness (mg/l as CaCO3)	LC_{50} (mg/l)	Reference
Rainbow trout (juvenile) *Salmo gairdneri*	sodium arsenate	FT	- -	10.8	Hale (1977)
Brook trout (adult) *Salvelinus fontinalis*	sodium arsenite	FT	140-152	14.9	Cardwell *et al.* (1976)
Goldfish (juvenile) *Carassius auratus*	"	FT	140-152	26.0	Cardwell *et al.* (1976)
Spottail shiner *Notropis hudsonius*	"	S	-	27.0	Boschetti and McLoughlin (1957)
Fathead minnow (juvenile) *Pimephales promelas*	"	FT	140-152	15.6	Cardwell *et al.* (1976)
Channel catfish (juvenile) *Ictalurus punctatus*	"	FT	140-152	18.1	Cardwell *et al.* (1976)
Channel catfish (fingerling)	"	S	-	15.0	Clemens and Sneed (1959)
Flagfish (fry) *Jordanella floridae*	"	FT	140-152	28.1	Cardwell *et al.* (1976)
Green sunfish *Lepomis cyanellus*	sodium arsenate	S	-	150.0	Sorenson (1976)
Blugill (juvenile) *Lepomis macrochirus*	sodium arsenite	FT	140-152	41.7	Cardwell *et al.* (1976)
Bluegill (fingerling)	"	S	29	0.29	Hughes and Davis (1967)
Bluegill	"	S	53	15.3	Inglis and Davis (1972)
Bluegill	"	S	210	16.2	Inglis and Davis (1972)
Bluegill	"	S	365	15.8	Inglis and Davis (1972)

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[a] FT = flow through; S = static.

TABLE 6-2. ACUTE TOXICITY OF ARSENIC FOR FRESHWATER INVERTEBRATES

Organism	Test Compound	Bioassay Method	Hardness (mg/1 $CaCO_3$)	LC_{50} (mg/1)	Reference
Cladoceran *Daphnia magna*	Sodium arsenate	S	45	7.4	Biessinger and Christensen (1972)
Cladoceran *Daphnia magna*	Sodium arsenite	S	-	5.3	Anderson (1946)
Cladoceran *Daphnia pulex*	"	S	-	1.0	Sanders and Cope (1966)
Cladoceran *Simocephalus serrulatus*	"	S	-	0.8	Sanders and Cope (1966)
Stonefly *Ptaronarcys californica*	"	S	-	22.0	Sanders and Cope (1966)
Scud *Gammarus pseudolimnaeus*	Sodium arsenite	FT	-	0.9	U.S. EPA (1980c)

on day 13. Five week-old fry exhibited 50% mortality at 2.6 mg/l; the LC_{50} for 12-week-old fry was 16.0 mg/l. Thus the tolerance of Muskellunge fry to arsenic increased rapidly as development progressed (Spotila and Paladino 1978).

The finding that the toxicity of trivalent arsenic to bluegills did not differ significantly over a range of $CaCO_3$ concentrations in water indicates that water hardness does not affect arsenic toxicity (U.S. EPA 1980a). Sorenson (1976) found that the time for 50% mortality to be reached in a group of green sunfish (Lepomis cyanellus) decreased with increasing temperature. Arsenic at 60 mg/l was toxic to 50% in 678 hours at 10°C and in 124 hours at 30°C. This was probably due to an increased rate of metabolism at higher temperatures. Specific studies on the effects of other water quality parameters on arsenic toxicity were not available. (Further discussion of the environmental factors and situations that favor availability of arsenic to aquatic organisms is found in the following subsection 6.2.)

In general, toxicity of arsenicals to fish increases with longer duration of exposure. The earlier life stages were usually more sensitive to arsenic than the juvenile or adult stages, and the early life stage toxicity values were also lower than the most sensitive invertebrate species tested (U.S. EPA 1980a).

6.1.3 Saltwater Biota

The data base for marine organisms is much less extensive than that for freshwater species and is limited to four fish and three invertebrate species (Table 6-3). The range in reported toxicity values for both arsenic trioxide and sodium arsenite to salmon, Atlantic silverside, stickleback, white shrimp, and shellfish, is quite narrow: 3,490 µg/l for bay scallops to 24,700 µg/l for juvenile white shrimp. Arsenic trioxide was found toxic to two species of salmon in the concentration range of 7,1200-12,300 µg/l. No data concerning chronic effects were available.

6.1.4 Phytotoxicity

As is the case for other species, the form of arsenic determines the element's adverse effects on plants. Sodium arsenite causes wilting and loss of turgor, leading to membrane deterioration in plants, apparently due to reaction with sulfhydryl enzymes (NAS 1977). The effects of arsenate are less immediate and less extreme, such as interference in the adenine diphosphate/adinine triphosphate process and interference with other plant enzymes (e.g., fumarase) (NAS 1977). Arsenic is an effective herbicide, applied usually as the trioxide or in organic form.

Phytotoxicity is correlated with available arsenic and not with total arsenic levels (NAS 1977). The amount of arsenic available in soil solution depends upon the degree of adsorption and precipitation of arsenic in soil, which is determined by the organic matter content,

TABLE 6-3. ACUTE TOXICITY OF ARSENIC FOR MARINE BIOTA

Organism	Test Compound	Bioassay Method[a]	Test Effect	Concentration (mg/l)	Reference
Chum salmon *Onchorhynchus keta*	Arsenic trioxide	FT	48-hr LC_{50}	8.3	Alderice and Brett (1957)
Pink salmon *O. gorbuscha*	Arsenic ion	-	96-hr LC_{100}	12.3	Holland *et al.* (1960)
Pink salmon *O. gorbuscha*	Arsenic ion	-	7-day LC_{100}	7.1	Holland *et al.* (1960)
Bay scallop *Argopectan irradians*	Sodium arsenite renewal	-	96-hr LC_{50}	3.5	Nelson *et al.* (1976)
American oyster (embryo) *Crassostrea virginica*	Sodium arsenite renewal	S	48-hr LC_{50}	4.3	Calabrese *et al.* (1973)
White shrimp *Penaeus setiferus*	Arsenic trisulfide	-	96-hr LC_{50}	24.7	Curtis *et al.* (1978)

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[a] FT = flow through; S = static.

the type and amount of clay present, the pH and the presence of other minerals. Aluminum and iron easily form complexes with arsenic; however, the presence of phosphorus may interfere with complexing (Woolson et al. 1971a; Woolson et al. 1971b). Discussion of types of soil and environments in which exposure of terrestrial plants is greater is found in Section 6.2.2.

Table 6-4 presents the results of phyotoxicity studies on various plant species, selected because they are likely to be consumed by humans and thus provide an exposure pathway. The large amount of literature regarding the efficacy of arsenicals as herbicides is not presented here; however, it has been summarized elsewhere (e.g., NAS 1977).

6.1.5 Microorganisms

The form of arsenic to which microbial populations are exposed is a factor of equal importance as in fish and mammal species. Both arsenate and arsenite are forms to which microorganisms would be exposed, with arsenate predominating in natural, aerobic waters and arsenite under reduced conditions such as those found in eutrophic lakes or in wastewater treatment. In fact, as described previously (Chapter 4.0) microorganisms themselves play an important role in the reduction of arsenate to arsenite. Arsenite is more toxic than arsenate (NAS 1977) and the effects levels of the organic form are generally one to two orders of magnitude lower than those for inorganic arsenic (Holm and Cox 1978). The toxicity of inorganic arsenic to microorganisms may be reduced by the presence of phosphate (DaCosta 1972).

Table 6-5 presents the results of laboratory studies investigating the effect of arsenic on microbial populations. The results are difficult to extrapolate to field conditions because most experiments were conducted under simple laboratory conditions or a special set of field conditions. In either case, the experimental design fails to replicate the complex behavior and transformations of arsenic in soil and water. Since key factors controlling the amount available for microbial uptake--absorption, leaching, methylation, and volatilization among others--are not represented, the effects levels reported are not very meaningful for environmental conditions.

Different compounds tested under the same experimental regime, however, can be compared. The relatively high toxicity of arsenite is attributed to its greater proclivity for binding to cell tissue, especially sulfhydryl groups (NAS 1977). The relationship between microbial conversion of arsenate to arsenite and the toxic effects on the mediating organisms that result from the formation of the more toxic form is not well understood. The organic arsenic, cacodylic acid, was found to be less toxic than inorganic forms. Peoples (1978) found arsenic in the trivalent form to significantly reduce mixed bacteria populations at 290 mg/l, while the pentavalent form had little effect at levels up to 10,000 mg/l.

TABLE 6-4. TOXICITY OF ARSENICALS TO TERRESTRIAL PLANTS

Form	Concentration	Effect	Source
Total arsenic (probably +5)	25-85 mg/kg (3-28 mg/kg soluble)	General critical level at which crop yield may be depressed	Walsh and Keeney (1975)
Total arsenic	44 mg/kg (6 mg/kg soluble)	Depressed growth in blueberry	Walsh and Keeney (1975)
Total arsenic	25 mg/kg	Depressed growth in snap beans, peas	Walsh and Keeney (1975)
MSMA DSMA	2.2-2.8 kg/ha 2.2-3.4 kg/ha	Controls growth of nutsedges (*Cyperus* spp.) and Johnsongrass (*Sorghum halepense*)	Hiltbold (1975)

TABLE 6-5. EFFECTS OF ARSENIC ON MICROORGANISMS

Species or Source of Population	Form of Arsenic	Concentration	Effects	Reference
Bacillus cereus	Arsenite	0.4 mM[a]	Growth inhibition	Mandel *et al*. (1965)
	Arsenate	10 mM[a]	Growth inhibition	
Nitrifying micro-organisms	Arsenate	0.1-1000 mg/l	At 100 mg/l decreased activity of *Nitrosomonas*; all concentrations decreased activity of *Nitrobacter*.	Holm and Cox (1978)
Nitrifying micro-organisms	Cacodylic acid	0.1-1000 mg/l	Only at 1000 mg/l was nitrification inhibited.	
Lake population	Arsenate and Arsenite	40 uM/l (~3 mg/l)	Organic matter degradation reduced by 50% during winter (low nutrient levels) but no effect during summer. The rate of phosphorus formation and algal uptake of arsenic were also reduced in the presence of both forms.	Brunskill *et al*. (1980)
Soil population (nitrifying microorganisms)	Arsenate and Arsenite	50 uM (in 3 ml solution applied to 10 g soil)	Minor inhibition of nitrogen mineralization by both forms.	Liang and Tabatabai (1977)

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[a] No concentration given.

6.1.6 Conclusions

Although comparable toxicity data are not available for all of the various arsenic compounds, there appears to be no significant difference in toxicity between trivalent and pentavalent inorganic forms for a given species. Organic arsenicals are generally less toxic than the inorganic forms. The range in values of arsenic toxicity for freshwater species encompasses over 3 orders of magnitude and is similar for fish and invertebrates. For example, acute toxic effect levels for aquatic biota range from approximately 290 μg/l (bluegill fingerling, sodium arsenite) to 150,000 μg/l (sodium arsenate, green sunfish). Most values are in the range 1,000-50,000 μg/l. The limited data available on saltwater biota were for the trivalent form only and indicated LC_{50}'s for invertebrates and fish between 3,500 μg/l and 25,000 μg/l. For many terrestrial food crops, a range of total arsenic between 25 mg/kg and 85 mg/kg (soluble arsenic from 3 to 28 mg/kg) was the critical level for depressed growth. Microorganisms were more sensitive to trivalent than pentavalent arsenic and to inorganic than organic forms. It is difficult to extrapolate laboratory-derived effects levels (from plate culture studies) to environmental conditions and no information on inhibitory levels of arsenic in natural soil or aquatic systems was available.

6.2 EXPOSURE OF NONHUMAN BIOTA

Terrestrial and aquatic organisms are exposed to arsenic, primarily in the form of arsenate and arsenite, via several environmental pathways. The specific chemical form involved is usually unknown as a result of the typical analytical practice of measuring total arsenic concentration. Nevertheless examples of characteristic exposure situations can be described from data presented previously in the Materials Balance (Chapter 3.0) and Fate and Monitoring sections (in Chapter 4.0). The following section describes exposure pathways and attempts to quantify exposure for aquatic and terrestrial populations on the basis of typical and maximum levels in surface water, likely sources of high exposure levels, and how the environmental fate of arsenic may affect the exposure of biota.

6.2.1 Aquatic Species

6.2.1.1 Limitations of Available Data

Most aquatic effects studies concern the effects of trivalent arsenic (arsenite). According to the previous discussion, there is less than an order of magnitude difference between lethal levels of arsenate and of arsenite based on the limited data available (see Table 6-1 and 6-2). Further, only limited monitoring data are available distinguishing between the different arsenic forms. Hence, though exposure to the various environmentally important inorganic arsenic can not be differentiated, the similarity in toxicity levels suggests that this is not a serious limitation. Organic arsenicals, however, are considerably less toxic than inorganic forms probably due to a low absorption efficiency (see Chapter 6.1). Again, however, monitoring data distinguishing organic from inorganic arsenic compounds are almost non-existent. Hence at best a qualitative judgement can be made of situations in which high levels of organic arsenic may be present.

6.2.1.2 Background Levels

Arsenic is present in natural waters at background levels of great variability ranging over six orders of magnitude. Reported natural concentrations in fresh waters from numerous sources have been compiled by the National Academy of Sciences (refer to Table 3-4, NAS 1977). These levels are generally within the range of surface water concentrations based on the data presented in Section 4.4 (Monitoring), ranging from 0.1 to >100,000 μg/l. In assessing the local impact of specific industrial discharges of arsenic, it would be ideal that the background concentration at that location be known in order to identify to what extent intentional releases of arsenic are responsible for exceeding natural background concentrations of the element in the vicinity of the release. In areas with already high natural levels, an additional release may have little incremental effect on concentrations affecting aquatic communities. Additionally, areas with historically high concentrations of arsenic are likely to be populated by arsenic-acclimated or resistant species, which may be unaffected by increases to concentrations that elicit adverse effects in bioassays on standard laboratory species. Unfortunately, data from pristine areas are few and even ensuring that a particular area is unpolluted is difficult because of the global nature of some intentional releases (e.g., to air). Therefore, distinguishing the additive effect of intentional releases in an area with natural background levels can be close to impossible.

6.2.2.3 Fate Considerations

In understanding exposure of aquatic species to arsenic it is not enough to know only the form and amount released. Upon the entry of arsenic to surface water, several fate processes may take place, which determine the availability and predominant form present for biotic exposure. The chemical form of the initial arsenic release and ecosystem parameters will both influence the equilibrium conditions following release. The following discussion attempts to describe some of the more common transformations that determine the exposure of aquatic communities.

On release, some fraction of the arsenic entering a lake or stream coprecipitates with hydrous oxides and partitions into the sediment. Apparently the pentavalent form is most likely to react in this manner; trivalent is oxidized to the pentavalent form in aerobic waters and then proceeds as described (Ferguson and Gavis 1972). The sediment to water ratio (for total As) is usually on the order of two to three orders of magnitude, as indicated by available monitoring data (see Chapter 4.4). Sediment high in available iron, aluminum, and manganese oxides is more likely to have high arsenic levels than other mineral or organic sediments.

Most of the arsenic present in sediment is stable, in the form of insoluble sulfides or as arsenic metal. Some of the arsenic, probably a small amount, may be subject to microbial methylation. One product of this activity, dimethylarsine, is more soluble than the previous forms and likely to move into the water column and be transformed to methanearsonic acid (MSMA). Other transformation products include cacodylates and trimethylarsine (NAS 1977).

Sediment will, therefore, act as a reservoir for waterborne arsenic, which would otherwise be available for uptake by most fish and algae. Accumulation in sediment, especially in the surface layer, may expose benthic organisms to high arsenic levels. Monitoring data (see Chapter 4.0, Pathway 3) indicate slightly higher tissue levels in organisms associated with the sediment layer than in those associated with the water column; the difference observed, however, was not great. The small difference implies either that uptake from sediment is not as efficient a process as from water (due to the chemical form present, mechanism of uptake and absorption efficiency, etc.) or that species differences in the ability to bioaccumulate are more significant than arsenic concentration differences between water and sediment.

Another implication of arsenic accumulation in sediment is the potential for subsequent release to the water column under conditions of turbulence or chemical change (e.g., pH, redox). Seasonal upwelling of bottom sediments, continual decreases in the pH of northern lakes due to influx of acid rain, and increased microbial activity can upset the equilibrium conditions governing the retention of arsenic in sediment. No specific incidences are known of fish kills or increased bioaccumulation due to environmental changes affecting availability, but the potential for such an occurrence must be recognized.

The arsenic discharged to freshwater that is not subject to removal by adsorption onto sediment will be present primarily in the pentavalent inorganic form or as an organic acid under buffered, aerobic conditions such as in streams, well-mixed lakes, and epilimnion (upper layer) of most static bodies of water. At least a small fraction of originally discharged trivalent will remain untransformed (Ferguson and Gavis 1972). Under reduced conditions, such as in some small stagnant ponds, eutrophic lakes, and in the lower reaches of deep lakes, the prevailing form is likely to be the trivalent. Distinction between these two forms in exposure and effects assessment does not appear to be as important for aquatic species as it is for human exposure. This is because of the similarity in the toxicity of the two forms for those aquatic species bioassayed.

Other chemical forms of arsenic, some highly soluble and available for biological uptake, are also present in the water column. Some of these include DSMA, MSMA, and cacodylic acid. The very limited monitoring data available on specific forms of arsenic indicate that concentrations of methylarsenic acids may sometimes exceed concentrations of arsenate and arsenite. Braman and Foreback (1973) found these organic arsenic forms, usually DSMA, to be the predominant species in three Florida aquatic systems (a river, pond and estuarine bay) out of the 10 systems sampled and to be equivalent to inorganic levels in three other ecosystems (see Table 4-7 in Chapter 4.0). More extensive monitoring efforts may indicate this to be a widespread occurrence in aquatic systems. The implications of this for exposure and risk are difficult to discern in the absence of effects data for each of these organic arsenicals. They are commonly described in the literature as being less toxic than inorganic forms; and the data available (on only monosodium methanearsonate--U.S. EPA 1980a) did support this statement.

Arsenic discharged to estuarine or marine waters that is not adsorbed onto sediment is found predominantly as arsenate. Oxidation of the trivalent forms will occur in aerobic areas, as occurs in freshwater. The reaction rarely goes to completion , however; the ratio of arsenate to total arsenic in ocean water was 0.8 (Sugawara and Kanomori 1964). The preceeding discussion on fate processes affecting arsenic in freshwater systems is generally applicable to saltwater systems as well.

6.2.1.4 Monitoring Data

STORET water quality data for ambient waters in major U.S. river basins indicate that 99% of the values for arsenic (36,308 observations) for the period 1975-1979, were less than 100 μg/l. Of these, 72% were in the range of 0.2 μg/l to 100 μg/l. Approximately 1% of the values (275 observations) were from 100.1 μg/l to >1,000 μg/l, a range that includes the total range of reported toxic effects levels to aquatic organisms (approximately 200-150,000 μg/l). Fifty-one observations were greater than 1,000 μg/l, which is approximately the threshold level for acute toxic effects to both freshwater and marine fish and invertebrates. The implications of this finding for the potential risk of aquatic communities is discussed in Chapter 7.

The Missouri River Basin, for which 22 of the observations in excess of 1,000 μg/l had been recorded, was examined in greater detail in order to determine the frequency and locations of these higher concentrations. Station data of mean values greater than 10 μg/l are presented in Table 6-6. All stations were from minor river basins 901 and 904 within the Missouri major basin. The means exceeding 500 μg/l were generally clustered in southwestern South Dakota and at a mine site near the town of Arrow Creek, Montana. The 5-year mean levels for the entire minor river basins 901 (Upper Missouri R.) and 904 (Central Missouri R. and Cheyenne R.) were 42 μg/l (198 observations) and 461 μg/l (473 observations), respectively. The sources of high levels in South Dakota are not known. There are no copper, zinc, lead, aluminum on other metals mines or smelters in the vicinity; however two large coal burning power plants are located in the western part of the state (JRB 1980). It is also possible that natural geological deposits of arsenic may be

TABLE 6-6. MONITORING STATIONS IN UPPER MISSOURI RIVER BASIN REPORTING HIGH MEAN ARSENIC CONCENTRATIONS (1975-1979)[a]

Station	Mean Arsenic Concentrations (μg/1)	No. of Observations
Montana (Minor River Basin 901)		
Galena Creek just below mine dump	543	12
Silver Creek at road above mouth	37	12
Anacondo Co., Great Falls Plant #1	14	2
Anacondo Co., Great Falls Plant #2	34	1
South Dakota (Minor River Basin 904)		
Boxelder Creek below Nemo (Black Hills)	32	2
Annie Creek at Highway 14A	40	15
Belle Fourche River below Confluence	1,889	9
Belle Fourche River above Confluence	20	5
Whitewood Creek at Crook City	5,258	9
Whitewood Creek at Deadwood	6,395	9
Whitewood Creek above Gold Run Creek	26	9
Whitewood Creek above Belle Fourche	5,664	9
Belle Fourche River near Elm Springs	2,230	18
White River at State Highway	23	3
Bad River near Ft. Pierre	37	11
White River near Oacoma	32	19
Cheyenne River at Cherry Creek	38	20
North Dakota (Minor River BAsin 904)	38	20
North Fork Grand at Haley	82	13

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[a] These stations are those that had mean total As levels in water from 1975-1979 in excess of 10 μg/1. A total of 19 stations were reported in Minor River Basin 901 and 59 in Basin 904.

Source: STORET (U.S. EPA 1980b).

the source. In Montana, the high levels appear to be associated with the mine site.

Two other major river basins, the Upper Mississippi and the Colorado, also had observations of arsenic concentrations exceeding 1,000 µg/l over the past 5 years. A total of 11 and 10 such observations, respectively, were reported during the 5-year period. The minor river basins recording the high levels were not examined in greater detail in this analysis; however, more thorough consideration at some future time is warranted.

Very little monitoring data are available for arsenic levels in marine waters. Concentrations in seawater are believed to be fairly constant, at a range of about 1-4 µg/l (LeBlanc and Jackson 1973). According to the data for arsenic residues in fresh and marine aquatic species, there is little difference between total body tissue levels in animals from the two types of water (NAS 1977).

6.2.1.5 Exposure Near Direct Sources of Arsenic to Surface Water

Introduction

Arsenic enters surface water directly through purposeful industrial discharge or herbicidal application. Inadvertent sources occur during fossil fuel combustion and production of other elements. According to the Materials Balance (Chapter 3.0), approximately 5% of all environmental discharges are made directly to surface water. The majority, 81%, is released to land with 11% to air. From both compartments, arsenic is transferred to surface water through, respectively, leaching and/or runoff and fallout (see Chapter 4.0). The total fraction of arsenic releases that eventually reaches water is unknown due to variations in form, environmental conditions, climate, and other factors.

The most significant estimated direct dischargers of arsenic to surface water in order of decreasing magnitude (annual rates) are pesticide users (aquatic and terrestrial applications), zinc and phosphorous production operations and fossil fuel combustion. Production of various other elements and alloys had lower associated annual releases. Pesticide producers are also expected to release arsenic to surface water; however no information on the magnitude of these discharges was available. The only producer of arsenic (trioxide), itself, had a low estimated aquatic discharge. Exposure levels and likelihood of exposure to arsenic from these specific sources are described in the following subsection. In regard to industrial effluents, especially those described above, a complicating factor is the presence of other toxic substances in the discharge such as other metals and chlorinated hydrocarbons. These additional substances can contribute to the effluent's effect on the receiving system, depending upon their concentrations and synergistic effects.

Production Sites

The sole U.S. producer of arsenic trioxide, ASARCO, was estimated to discharge less than 1 kkg arsenic to surface water annually (see Chapter 3.0). All of the 1,200 kkg of solid waste produced annually was assumed to be land-disposed rather than deposited in water, in order to examine the local effects of these releases. The Pacific Northwest Major River Basin in which the ASARCO plant is located was examined and found to have levels exceeding 1,000 μg/l (total arsenic) in surface water only two times in the last 5 years according to the available STORET data (U.S. EPA 1980b). Examination of minor river basin 1311, Puget Sound, in which the plant is found indicated that arsenic levels have not exceeded 10 μg/l at any of the 45 stations for the past 5-year period. Mean levels for the entire minor river basin for both remarked (4 observations) and unremarked data (49 observations) were approximately 1-3 μg/l. These results are compatible with the low reported industrial discharge rate.

The possibility that a significant fraction of the arsenic released into Puget Sound was absorbed onto the sediment was also examined. Sediment data for the Pacific Northwest, however, did not show significantly higher levels of arsenic than in other parts of the country. This may reflect averaging effects due to aggregating a large amountof data. However, other explanations can be that the amount of arsenic discharged to water in the Pacific Northwest basin is not significant enough to be reflected in sediment levels or that sediment transport out of the system is rapid enough to keep arsenic levels in the sediment layer diluted. The few observations of arsenic levels in water in the minor river basin downstream from the ASARCO plant were at levels even lower than the Puget Sound data; however the small sample size precludes any conclusions.

Use as Aquatic Herbicide

Sodium arsenite was used in the past in applications directly to surface water at concentrations of approximately 10 mg/l (Ferguson and Gavis 1972) to control aquatic vegetation. It is not known how common its use is in the U.S. at this time, although any use is probably in southern regions where vegetative growth in waterways is more of a problem than in colder areas. The total amount used annually is estimated at less than 90 kkg (JRB 1980); thus on a national scale use is not very significant compared to terrestrial applications of other arsenicals. There is a potential for long term persistence of arsenic in the sediment of treated lakes with subsequent release during turbulence or mixing (see Chapter 4.0 Pathway 5).

Runoff to Surface Water From Pesticide Use on Land

The majority of arsenic produced is used in pesticides. Runoff to surface water from land application of arsenicals is expected to account for 7% of the total amount of pesticide use each year according to measurements made in Texas watersheds (Richardson et al. 1978).

This results in a loading of 720 kkg of arsenic annually. Runoff would be greatest in areas with high rainfall rates, from poorly managed lands and in areas in which arsenic use is concentrated. Approximately 85% of both DSMA and MSMA use is in the southeast and south central regions of the U.S. (JRB 1980). Arsenic acid use is predominately in Texas, and Oklahoma, both states in the south central region (JRB 1980). No information was available on the distribution of use of cacodylic acid; however since it is primarily for lawn restoration, most use is probably in more Southern regions. The distribution of use of other arsenical pesticides is not known. The southeastern region of the U.S. has a high rate of soil erosion due to high clay soils, poor management techniques and a high rainfall rate. It is expected that entry of arsenic to surface water through pesticide runoff (at least of compounds described above) is greatest in the Southeast and possibly the South Central regions of the U.S.

Much of the arsenic carried in runoff is expected to be adsorbed onto soil particles in the form of the pesticide and to eventually settle into the bottom sediment layer of the receiving aquatic system. Some arsenic is likely to go into solution on contact with water and become part of the system's cycling of arsenic as described in Section 6.2.1.5.

Other Releases

Aquatic releases from mineral mining and processing are from acid mine drainage and process wastewater discharges. Arsenic is released also in discharges of phosphorus-containing detergents to surface water. Surface water discharges from fossil fuel combustion originate from acid mine drainage and from fly ash ponds, both in effluents and from overflows and leaks (see Chapter 3.0). Arsenic levels (total As) from 0.001 mg/l to 4.8 mg/l have been measured in zinc processing wastewater effluents (Appendix A, Note 11). Concentrations of arsenic in washwater are on the order of 0.15 mg/l to 250 mg/l (see Section 5.2.3). Concentrations of 12 mg/kg in acid mine drainage and 15 μg/l to 60 μg/l in disposal pond effluents have been reported (see Appendix C). All of these wastestream concentrations are low enough that, following dilution on discharge, it is unlikely that significant levels in water would result from sporadic releases. Where continuous industrial discharges are made or in mine drainage areas, there is a possibility that high arsenic levels would build up in sediment and water.

A field study found little environmental impact of arsenic-bearing mine drainage on a small harbor in Newfoundland (Penrose et al. 1975). Arsenic concentrations were elevated above background levels only within 100 m from the mine, sediment levels only within 50 m. Out of the aquatic biota sampled, only sea urchins showed higher than background levels in their tissues.

No other field studies were available describing the impact of these releases in natural ecosystems. Therefore, any conclusions made about the significance of these arsenic sources are, at best, speculative.

6.2.2 Terrestrial Biota

Terrestrial systems receive the largest fraction (81%) of the total environmental releases of arsenic. There is a strong possibility that terrestrial biota -- plants and microorganisms -- will be exposed to high levels of arsenic. Monitoring data indicate high levels of arsenic (total As) in excess of 10 mg/kg in soil associated with industrial or agricultural activities and also in naturally contaminated areas. Table 6-7 lists some of the reported soil levels. The highest concentrations are associated with orchards which received applications of lead arsenate in the past. Although current use of these pesticides is greatly reduced, the residues already accumulated are very persistent.

There appears to be a relationship between the degree of phytotoxicity and plant bioaccumulation and the concentration of soluble arsenic in the soil solution (see Section 6.1.4). Therefore, concentrations of total arsenic in soil listed in Table 6-7 overestimate the amount of the element actually available for uptake. Monitoring data reporting soluble arsenic levels, however, are uncommon.

Regardless of the form initially released, it is expected that arsenate will be the predominant form of exposure in the upper aerobic layer of soil which supports plant growth and microbial activity. As described in Chapter 4.0, the ratio of arsenate to arsenite is usually 8 or 9:1; sometimes DSMA is present at levels comparable to those of arsenite. Microflora in flooded soils and trees with deep roots may be exposed to higher levels of trivalent arsenite than those usually measured in the soil surface layer.

The discussion concerning potential contamination of ground water by arsenic presented in Section 5.2.2.1 is also applicable to the potential for exposure of terrestrial communities. In both cases the soluble, mobile fraction of arsenic is of importance in ultimate exposure. Table 5-5 describes the geographic distribution of known arsenic releases to land. The most significant sources are fly ash disposal ponds, smelting plants and use of arsenical pesticides. These sources tend to be concentrated in the industrial northeast and central states. The distribution of natural high levels of arsenic is unknown.

Two factors probably minimize the potential for exposure of certain terrestrial species and communities. Plants which are of economic importance or significant as human exposure routes are generally not cultivated in the vicinity of industrial operations. Additionally. the use of arsenical pesticides on food crops is restricted. Therefore, exposure of economically-important plants to high arsenic levels is not likely. Also there is little evidence for significant lateral movement

TABLE 6-7. EXAMPLES OF TERRESTRIAL SITES OF SIGNIFICANT ARSENIC EXPOSURE[a]

Location	Concentration in soil
Vicinity of fly ash disposal ponds	6.5 mg/kg[b]
Downwind from copper smelters: Montana and Washington	150-380 mg/kg
Orchards receiving arsenical pesticide treatment	165-627 mg/kg maximum 2,500 mg/kg
Potato fields receiving arsenical pesticide treatment	6-28 mg/kg
Soils with high natural background levels	up to 80 mg/kg although usually < 10 mg/kg

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[a] These data were measured in soil at specific sites and are not meant to be representative of all situations.

[b] Limited data. Higher levels may exist.

Source: Chapter 4.0.

of arsenic in soil with the exception of areas with significant wind erosion (see Chapter 4.0). Most migration tends to be downward, away from the soil layer supporting terrestrial life. Therefore, the exposure of natural communities of plants and microorganisms to high anthropogenic arsenic levels is expected to be concentrated in the immediate vicinity of specific sources.

6.2.3 Conclusions

Aquatic species are exposed to numerous chemical forms of arsenic, but the limitations of the monitoring data base prevent consideration of specific forms other than arsenate, arsenite and several organic arsenical compounds. Natural background levels of arsenic in surface water are similar in magnitude and variability to arsenic levels resulting from human activities. Ideally, the background concentration at a particular location should be known in order to assess the impact of an intentional discharge on aquatic communities; in practice, this information is difficult to obtain. According to data on the chemical speciation of arsenic in aquatic systems, a large fraction of discharged arsenic accumulates in the bottom sediment, associated with iron, aluminum, and manganese hydrous oxides. These concentrations are commonly two to three orders of magnitude higher than concentrations in the water column. Sediment-burrowing and other benthic species would be exposed to these concentrations. Most fish species are exposed to the remaining fraction of arsenic in the water column, and usually at total arsenic levels lower than 100 µg/l. Arsenate is thought to be the predominant form under aerobic conditions and arsenite under anaerobic conditions. Limited monitoring data indicate that organic species, particularly DSMA, may be the major form in certain water bodies.

Approximately 50% of the observations exceeding total arsenic levels of 1000 µg/l over the past 5 years occurred within the Missouri major river basin. These high concentrations occurred primarily in southwestern South Dakota and did not appear to be directly associated with any particular industrial site. High levels were also observed in Montana in the vicinity of a mining site. The remaining levels exceeding 1000 µg/l were reported in the Upper Mississippi and the Colorado major river basins.

Potential industrial or use-related sources of arsenic releases affecting aquatic communities include arsenic production sites, aquatic herbicide application, runoff from application as a terrestrial pesticide, mining and metals-processing facilities, fossil fuel combustion plants and discharge of phosphorus-containing detergents. Although the total contribution from some of these sources, particularly pesticide runoff, metals production, and detergent discharges, is significant on a national scale, the concentrations detected in effluents are not high (usually < 13 mg/l) according to the limited data base available. Virtually no information was available regarding the long-term impact of continual arsenic releases on aquatic communities in terms of productivity or diversity.

Terrestrial communities of plants and microorganisms may be exposed to high soil arsenic levels in excess of 100 mg/kg (total As) in the vicinity of smelting operations and in areas receiving arsenical pesticide treatment. Also natural background levels in soil may be as high as 80 mg/kg, although they are usually less than 10 mg/kg. Since the soluble fraction of arsenic in soil is what is most important in terms of terrestrial exposure, monitoring data (reported as total As) tend to overestimate the amount of arsenic actually available for uptake. High arsenic concentrations are expected to be usually confined in the immediate vicinity of arsenic sources.

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7.0 RISK CONSIDERATIONS

7.1 RISK CONSIDERATIONS FOR HUMANS

7.1.1 Introduction

Arsenic exposure via inhalation, dermal contact, and ingestion poses a potential risk to human health. This section focuses on the risk associated with exposure via water-borne pathways through ingestion or dermal contact. Inhalation of arsenic from air-borne pathways, as discussed in Chapter 5.2 has been the specific focus of other investigators (see Suta 1980; CAG 1978). Therefore inhalation is only briefly considered in this chapter, primarily to put the exposure levels of other routes in perspective.

An objective of the risk assessment process is the quantification of risks to various human subpopulations. Quantifying such risks requires:

- Careful identification of the subpopulations at risk and the populations exposed (Section 5.2);
- Evaluation of the ranges of exposure for each subpopulation (Section 5.2);
- Determination of the effects levels or dose/response data in the species of concern and/or in proxies for these species (for example, laboratory animals as a proxy for humans; human epidemiological data; Section 5.1); and
- Extrapolation of dose/response data and determination of margins of safety for the subpopulations at risk (this chapter).

In this section, effects data are combined with exposure data in two ways:

- Effects levels observed in humans and laboratory animals are compared with human exposure levels; this procedure is followed for two inorganic forms of arsenic (arsenite and arsenate) and for organic arsenicals in general. Also exposure levels for total arsenic in various combinations of exposure pathways (exposure scenarios) are compared with effects data.
- The magnitude of human risk is estimated with respect to arsenic levels in drinking water that may cause adverse effects based on the Taiwanese epidemiologic data on skin cancer (calculated in Chapter 5.2); trivalent and pentavalent forms of inorganic arsenic

are considered together in this estimate because of the lack of adequate monitoring data distinguishing between them.

Throughout the previous chapters of this report, the importance of the chemical form of arsenic in determining chemical fate, effects and magnitude of exposure has been stressed. Because of this dependence, risks associated with arsenic exposure are evaluated as best as possible for the different chemical forms of arsenic. The most significant chemical forms in terms of environmental abundance and availability of information on human health effects, fate, and exposure are the trivalent and pentavalent inorganics and organic arsenicals (primarily MSMA, DMSA, cacodylic acid). Due to the infrequency of their environmental occurrence or to lack of data requisite to conduct a risk assessment, all other arsenic compounds are grouped into a fourth category and discussed only briefly. The differentiation of chemical forms of arsenic is maintained throughout the risk discussion to the extent possible. However, humans are generally exposed to a combination of different arsenic entities rather than to a single chemical entity, so that an epidemiologically-based risk estimate cannot attribute effects to the separate forms and the other estimates based on laboratory data, are, at best, approximate.

7.1.2 Effects of Arsenic

The human and mammalian health effects associated with arsenic exposure are discussed in detail in Section 5-1. Table 5-2 presented a tabulation of effects and associated reported levels, grouped by type of effect, and identified the chemical form where possible. Not all environmentally important forms are well represented among available laboratory toxicity studies for each exposure route. For example, virtually no laboratory data are available on the toxicity of arsine forms, which are nonetheless commonly reported as the most lethal arsenic form to humans. Of the eight studies available that quantify carcinogenicity and teratogenicity, seven concerned arsenate and only one concerned arsenite. All mutagenic studies available were for arsenate. The adverse effects reported for humans concerned arsenate (1 study), arsenic trioxide (1 study) and general arsenic (form unknown). Data on the organic arsenic forms were limited primarily to metabolic studies which in general indicated limited uptake of these forms but did not directly measure toxicity. The lack of a definitive and consistent data base for all chemical forms of arsenic is notable.

In various laboratory studies, low-concentration feeding experiments with arsenic compounds were negative with respect to induction of cancer. Teratogenic effects in mice have been associated with ingested sodium arsenate at a daily dose of 120 mg/kg and at lower levels when administered through intraperitoneal injection. Injection is not a realistic human exposure route for arsenic and is more appropriately used for comparison of species' differences. The positive mutagenic effects elicited by sodium arsenate in cell cultures are not in a form to allow comparison to exposure levels. Effects levels for humans have been derived primarily from episodes of accidental exposure to arsenic through ingestion of contaminated food or water. The effects levels listed on Table 5-3 were converted to mg/kg/day dose equivalents utilizing standard weight values and are presented in Table 7-1. Caution should be used in comparing different animal species, concentrations for different chemical forms of arsenic (i.e., the amount of arsenic comprising a concentration of Na arsenate will be greater than in Pb arsenate), and also different exposure routes.

Examination of this table indicates that the lowest chronic dose to induce an effect in humans (i.e., skin cancer) is that obtained from drinking water in Taiwan; peripheral neuropathy has also been reported with ingestion of 0.04 mg/kg/day of calcium arsenate for 2-3 weeks. The Taiwan study, based on the consumption of arsenic for more than 40 years by a study population of 40,421 individuals, provides the best data for use in risk extrapolation (in Section 7.1.6). The results of the study, however, must be attributed to all forms of arsenic present and related to the combined effect of their concentrations, assuming that the presence of substances other than arsenic did not contribute to the observed effects.

It is worth noting that the concentrations eliciting adverse effects in humans after short-term exposure are generally 5 to 10 times lower than the concentrations associated with no effects in long-term feeding studies with rats. However, the rat is unique among mammals in that 80-90% of the metal is bound to the globin portion of hemoglobin rather than uniformly distributed to body tissues. This difference may account for differences in concentrations needed to produce an effect in this species.

7.1.3 Exposure to Arsenic

Section 5.2 described the pathways leading to human exposure to arsenic. Table 7-2 summarizes the estimated exposure levels presented as arsenic per kg body weight (for a 70-kg human) per day. Ingestion and dermal contact were considered more thoroughly than inhalation exposure due to the existence of detailed exposure assessments for airborne routes of arsenic exposure (especially Suta 1980). Both food and water are believed responsible for at least low levels of arsenic, usually 0.1-0.3 μg/kg/day for a large fraction of the U.S. population. In subpopulations either drawing their water supply from wells in areas contaminated with high levels of arsenic or consuming highly contaminated fish, exposure levels can be much higher, as much as an estimated 167 μg/kg/day. Most (∿90%) of the unbound inorganic fraction of arsenic in water is absorbed in the digestive tract. Arsenic bound to biological tissue (e.g., in fish) is thought to pass through the gut largely unchanged. The lower levels of "free" arsenic present in the fish would probably be absorbed. Unfortunately, little information is available on the concentrations of these absorbable forms of arsenic in other kinds of food.

TABLE 7-1. ADVERSE EFFECTS OF ARSENICALS ON MAMMALS EXPRESSED IN DOSE EQUIVALENTS[a]

	Chemical Form	Effect/Species	Effects Dose Equivalent	No Apparent Effects Dose
Arsenic	unspecified	carcinogenicity in humans	0.05 mg/kg/day[b]	--
	unspecified (in milk)	CNS damage in humans (infants)	0.8 mg/kg/day[c]	--
	unspecified	lethal to humans (infants)	7.9 mg/kg/day[c] (for 1 month)	--
Arsenite	Na arsenite	carcinogenicity in rats	--	12.5 mg/kg/day[d]
Arsenic Trioxide		hyperkeratoses and hyperpigmentation in humans	0.13 mg/kg/day	--
Arsenate	Ca arsenate (in soy sauce)	peripheral neuropathy in humans	0.04 mg/kg/day (for 2-3 weeks)	--
	Na arsenate	carcinogenicity in rats	--	23 mg/kg/day
	Pb arsenate	carcinogenicity in rats	--	92.5 mg/kg/day
	Ca arsenate	carcinogenicity in rats	--	33.3 mg/kg/day
	Na arsenate	teratogenicity in mice	120 mg/kg (single oral dose)	--
	Na arsenate	teratogenicity in mice	10 mg/kg (single intraperitoneal injection)	--

[a] All data derived from summary Table 5-1 in Section 5.1.

[b] Assuming a 70-kg human and highest well water concentration.

[c] Assuming a 4.5-kg infant.

[d] All rat data based on consumption of 15 g feed/day in a 300 g rat.

TABLE 7-2. SUMMARY OF ESTIMATED ARSENIC EXPOSURE LEVELS ASSOCIATED WITH INDIVIDUAL EXPOSURE ROUTES

	Arsenic Intake (μg/kg/day)			
Ingestion	Form	Typical	Maximum	Absorption Coefficient[a]
1. Drinking Water				
Surface	+5	0.1	2.9	0.9
Ground Water	+3, +5, arsine?	0.1	57.1	0.9
2. Food				
Total Diet	all forms	0.3	2.7	1.0
High Fish	0, +3, others	14.3	142.9	may be very low
Moonshine	unknown	<0.01	5.9	0.9
Wine	+3, low +5	<0.01	7.1	0.9
3. Soil	+3	<0.01	0.3	1.0
Dermal Contact				
1. Handling Arsenic-treated Wood	unknown	3.4		already included[b]
2. Use of Arsenic-contaminated Detergent	+5		57[c]	already included
Inhalation				
1. Atmosphere				
General Urban	+3	<0.01	0.01	1.0
Vicinity of Sources	+3	0.1	0.3	1.0
Arsenic-treated House	unknown	<0.01-0.03		1.0
2. Cigarette Smoking	+3		1.3	0.35
3. Handling, Sawing, etc. with Arsenic-treated Wood	Unknown	3.7		already included

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[a] Assumed to be equal to 100% if no specific data available.

[b] An absorption coefficient is factored into the calculation of these exposures by the Special Pesticides Program, OPTS, USEPA.

[c] Based on dermal absorption rate of 23.2 mg arsenic/hour, which was measured at high arsenic concentrations. Rate may be lower at low arsenic concentrations. This estimate has a high degree of uncertainty.

NOTE: The sources of these levels and their uncertainties are discussed in Chapter 5 of this report.

Exposure to arsenic through dermal contact may occur with absorption from water, use of arsenic-containing pesticides, handling of treated wood, application of arsenic-containing medication, and use of arsenic-contaminated detergents.

7.1.4 Risk of Exposure to Different Forms of Arsenic

By comparing effects levels for the different forms of arsenic with exposure levels, ratios of exposure to effects levels can be calculated and compared and some conclusions drawn about the differences in risk different forms of arsenic pose for humans.

7.1.4.1 Arsenate

According to Table 5-11 (in Section 5.2) humans are typically exposed to about 0.1 μg/kg of arsenic daily, presumably in the form of arsenate, in surface drinking water. Exposure to arsenate in the total diet is expected to be much lower than the 0.4 μg/kg level estimated for total arsenic. No particular type of food is known to be contaminated primarily with the arsenate form. These levels are well below the no apparent effects levels based on laboratory animal studies and the epidemiological study presented in Table 5-1.

The exposure level (maximum) estimated for dermal absorption through use of arsenic-contaminated detergents was 57 μg/kg. No effects levels for the route of dermal contact were available for comparison.

Also, if the ground water level of 4,000 μg/l arsenic described as a maximum level of exposure in Table 7-2 is assumed to be 100% arsenate and consumed daily, this level exceeds the lowest reported concentration that was linked with skin cancer in Taiwan. However, the presence of appreciable levels of arsenite in the Taiwanese ground water was thought to be an important factor in the development of skin cancer in that population.

7.1.4.2 Arsenite

Even if 100% of arsenic in ground water is arsenite, the majority of the U.S. population is exposed to arsenite levels that are well below effects levels from laboratory studies for either arsenite or total arsenic. The epidemiological extrapolations from the Taiwanese study, however, estimate a higher incidence of skin cancer associated with even these levels. Due to the great uncertainty regarding this conclusion, these extrapolations are discussed separately in detail in 7.1.6.

Two other exposure pathways resulting in uptake of high arsenite levels are ingestion of highly contaminated seafood and wine (maximum level). The estimated levels exceed the effects level of 130 μg/kg/day for arsenic trioxide, which resulted in non-lethal skin disorders.

7.1.4.3 Organic Arsenicals

No information applicable to humans was available concerning the specific toxicity of the organic arsenicals. In general, however, these compounds (MSMA, DSMA, cacodylic acid) are less toxic than the inorganic forms of arsenic (see Chapter 5.0). In addition, metabolic studies on organically-bound arsenic in ingested foods indicate minimal absorption of arsenic. Therefore, the effects levels for total arsenic provide a very conservative estimate of toxicity for the organic arsenicals, as a first approximation.

Exposure pathways associated with potentially high levels of organic arsenicals include ingestion of some ground and surface water supplies, food, especially seafood, and soil. With the possible exception of diets high in contaminated fish and consumption of drinking water highly contaminated with organic compounds of arsenic, most pathways have exposure levels lower than the reported effects levels for total arsenic. The Taiwan epidemiological study did not report the presence of any organic arsenicals in water samples so it does not provide applicable effects level in this case.

7.1.5 Risk in Regard to Combined Exposures to Total Arsenic for Selected Subpopulations

The exposure levels derived in Section 5.2 for individual human exposure pathways were aggregated in order to estimate combined exposure levels, or exposure scenarios, for a number of subpopulations. Table 7-3 lists the exposure levels for water, assorted foods or general diet, and several inhalation and dermal exposure routes. Different routes of exposure are combined for an estimate of one total exposure level for purposes of comparison of different subpopulations. However, in assessing the significance of these combined levels in terms of human toxicity, the three exposure routes (ingestion, inhalation, and dermal absorption) must be disaggregated and compared with effects levels representing each particular route. Also included on Table 7-2 are absorption efficiencies for each route, which were reported in Section 5.1 and a value for exposure level expressed as a dose in μg per kg of body weight. This final value facilitates comparison with effects levels in Table 7-1.

7.1.6 Risk Extrapolations for Arsenical Skin Cancer

7.1.6.1 Introduction

The potential carcinogenic risk to humans due to ingestion of arsenic compounds is estimated below.

Ideally, this would be accomplished in two ways:

TABLE 7-3. SELECTED ARSENIC EXPOSURE SCENARIOS FOR SPECIFIC HUMAN SUBPOPULATIONS[a]

Exposure Medium	Exposure Scenarios (μg/day)									
	General Population	Smokers General Population	Child	High Fish Consumption	High Wine Consumption	Woodworker	High Activity Woodworker	Contaminated Well Water Consumption	Worst Case	Maximum Fish Consumption
Water	5	5	2.5	5	5	5	5	1,000	1,000	5
Food/Fish	21	21	11	21 + 1,000	21	21	21	21	21 + 1,000	21 + 10,000
Wine/Moonshine	0.01	0.01	-	-	500	-	-	-	500	-
Air	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	6	0.06
Cigarettes	-	90	-	-	-	-	-	-	90	-
Soil	-	-	20	-	-	-	-	-	20	-
Exposure to Wood	-	-	-	-	-	260	500	-	260	-
TOTALS	26	116	34	1,026	536	286	536	1,021	2,897	10,026
Per kg/day	0.4	2	8	15	7	4	7	15	41	143

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[a] The sources of these these levels and their uncertainties are discussed in Chapter 5 of this report.

1) Various extrapolation models would be applied to exposure data for occupational and general environmental exposures (from retrospective studies) in order to obtain an approximate dose/response relationship.

2) These same models would be applied to data from controlled experiments on laboratory animals, and the animal dose/response relationship converted to an estimated human dose/response.

In the first approach, the overriding uncertainty is in the data themselves: usually the exposure levels, lengths of exposure, and even response rates (responses per number exposed) are "best estimates," and furthermore, unknown factors (background effects, etc.) may bias the data. In the second approach, the data are usually more accurate, but the relationship between animal and human response rates is controversial. If one can perform both analyses and the results corroborate each other, confidence is gained in these results. If, on the other hand, data are not available for one of the analyses, and some result is assumed to be better than no result, the analysis must be based on what data are available.

Further complicating the issue is that presently there is no basis for judging the relative merits of the various extrapolation models. It is impossible to say which, if any, of them is correct. However, the models applied here are believed to bound the true risk, and are probably conservative (i.e., overestimate the risk).

7.1.6.2. Data and Discussion

The available data concerning effects on humans and other mammalian species were discussed in Chapter 5.

For arsenic, the only extensive quantitative carcinogenicity data currently available are from the studies by Tseng *et al*. (1968) of Taiwanese villagers exposed to arsenic through their drinking water between 1910-1920 and 1956-1966. (This study was discussed in Section 5.1.) The data from these studies relevant to this analysis are given in Tables 7-4 and 7-5: the incidence of skin cancer among villagers (categorized according to age and sex) exposed to various levels of arsenic in their drinking water.

Table 7-6 shows the chemical constituent makeup of a sample of the drinking water. The ground water in the area is expected to be anaerobic and some oxidation probably occurred between extraction and sampling (Irgolic 1979). Since no special precautions were taken to preserve the makeup of the sample during shipment from the collection site to the laboratory (in the U.S.), these figures are suspect. Nevertheless they are the best available at present. Based upon these

TABLE 7-4. AGE- AND SEX-SPECIFIC SKIN CANCER INCIDENCE RATE FOR STUDY AREA IN TAIWAN

	% Incidence (± 0.5)		
Age	Male	Female	Total
0	0	0	0
10	0	0	0
20	0	0	0
30	1	0	1
40	2	1	1.5
50	5.5	2	3.5
60	10	4	7
70	15.5	6	10.5

Source: Tseng *et al*. (1968)

TABLE 7-5. SKIN CANCER PREVALENCE RATE PER THOUSAND POPULATION, BY AGE AND ARSENIC CONCENTRATION IN DRINKING WATER, TAIWAN STUDY

Concentration Range (mg/l)	Age Group			
	20-39	40-59	60 and Over	Total
≥0.60	11.5	72.0	192.0	21.4
0.30 - 0.59	2.2	32.6	106.2	10.1
0.00 - 0.29	1.3	4.9	27.1	2.6
Undetermined				

Source: Tseng *et al*. (1968)

TABLE 7-6. CHEMICAL CONSTITUENTS OF TAIWAN WATER SAMPLES[a]

Chemical	Chemical Constituents (mg/l) by Geographic Location	
	Pei Men	Pu Tai
Arsenite[b]	.05	.09
Arsenate[c]	.52	.63
Sum	.57	.72
Total Arsenic (AAS)[c]	.72	.76
Total Arsenic (NAA)[d]	.76	--
Sodium	282	223
Copper	<.1	<.1
Manganese	<.1	<.1
Zinc	<.1	<.1
Iron	<.1	<.1

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[a] Also detected in some samples was an alkaline hydrolysate of ergotamine, lysergic acid, or similar compounds (Lu *et al*. 1977a).

[b] GC-MES.

[c] Flameless Atomic Absorption Spectrometry.

[d] Neutron Activation Analysis.

Source: Irgolic (1979)

data, a risk estimate is performed assuming that all arsenic compounds contribute equally to carcinogenic risk.

In contrast to the Tseng study, a study by Morton et al. (1976) showed no evidence of increased cancer incidence in Lane County, Oregon, an area in the U.S. where significant amounts of arsenic have been reported in the drinking water. This is possibly due to the lower concentration of arsenite in the Lane County water supply than in Taiwan, population shifts in the county, and lower duration of exposure.

In order to deal with the uncertainties inherent in extrapolation, three commonly used dose/response models have been applied to the data in Table 7-4 in order to establish a range of potential human risk. It must be emphasized that the assessment of potential human risk is subject to important qualifications:

- There have been both positive and negative findings for the carcinogenicity of arsenic in humans based on epidemiological studies.
- Due to inadequate understanding of the mechanisms of carcinogenesis, there is no scientific basis for selecting among several alternative dose/response models that yield widely differing results.
- The latency period for development of skin cancer is unknown; Wagner (1973) estimates it to be between 2 and 40 years.
- Which of the arsenic compounds, if any, is the primary contributor to carcinogenicity is not known.
- Carcinogenic contaminants other than arsenic in the Taiwanese villagers' water supplies may be responsible for their high incidence of cancer. Several fluorescent substances were identified including an alkaline hydrolysate of ergotamine, lysergic acid, or a similar compound (Lu et al. 1977). These substances are highly neurotoxic and could potentially have contributed to the effects observed in the population drinking the contaminated well water. Unfortunately these substances were not quantified in the samples nor was a large number of water samples analyzed for them.

7.1.6.3 Estimation of Human Risk

The concern here is with the probability of contracting cancer at some unspecified time during a lifetime, due to ingestion of arsenic at some previous time. Unfortunately, just as there is no basis for choosing among the various risk extrapolation models, so there is no clear procedure for taking into account the time lag between exposure and response (i.e., the latency period), and the models are not sophisticated enough to attempt this estimate. Thus, the data in Table 7-5 for the groups of people ages 20-39 and 40-59 cannot be analyzed, since the percentage in each of these groups that might yet contract cancer from previously ingested arsenic, even if further exposure ceased, is not known.

The people making up the last group, however, (people ages 60 and over), are more likely to show a significant increase in skin cancer cases during their lifetimes because this group approaches a lifetime exposure. These data were used in the extrapolations, recognizing that the true incidence is confounded by the normal increase in skin cancer incidence with age. Table 7-4 shows that the cancer rate among men in this age group is about 1.5 times the rate in men and women taken together. Therefore, in order to arrive at a conservative risk estimate cancer incidence in the 60-and-over age group is multiplied by roughly 1.5.

In addition to the latency problem, the models cannot deal with ranges of exposure producing levels of response, particularly not with open-ended ranges such as "greater than 0.60 µg/l." The average exposures for the low and medium exposure ranges were chosen while 0.70 mg/l was selected as representative of the high range, and probably conservative. The derived "data" in Table 7-7 were also used for the extrapolations. The arsenite exposure is (.05/.76) ≅ .066 times the total arsenic exposure. This value is conservative for the ratio of arsenite to total arsenic, from Table 7-6.

The three dose/response models used to extrapolate human risk were the linear "one-hit" model, the log-probit model, and the multistage model. The latter is actually a generalization of the one-hit model, in which the hazard rate is taken to be a quadratic rather than linear function of dose. All of these models are well described in the literature, and a theoretical discussion may be found in Arthur D. Little (1980). The one-hit and multistage models assume that the probability of a carcinogenic response is described by

$$P(x) = 1 - e^{-h(x)}$$

where P(x) is the probability of response to dose x, and h(x) is the "hazard rate" function. The log-probit model assumes that human response varies with dose according to a log-normal distribution. Due to their differing assumptions, these dose/response models usually give widely differing results when effects data are extrapolated from relatively high doses to the low doses typical of environmental exposure.

For the linear one-hit model, the equation

$$P(x) = 1 - e^{-Bx}$$

TABLE 7-7. ESTIMATED CARCINOGENIC RESPONSE[a] IN HUMANS EXPOSED TO ARSENIC IN DRINKING WATER

Level of Arsenic in Drinking water (mg/l)		Carcinogenic Response	
Total Arsenic	Arsenite	Rate	Percent
0.70	.046	290/1000	29%
0.45	.030	160/1000	16%
0.15	.010	45/1000	4.5%

---

[a] These data are extrapolated from an epidemiological study of skin cancer incidence in Taiwanese villagers exposed to arsenic in their drinking water. There are a number of uncertainties in extrapolating the results of this study to U.S. populations due to the presence of other potential carcinogens in the drinking water, intermittency of exposure, inadequate understanding of the mechanism of carcinogenicity, conflicting studies, among other factors. These uncertainties are discussed in Chapter 5.0 and 7.0.

Source: Derived from Tables 7-4, 7-6.

is solved for the parameter B. From the data for arsenic discussed previously, B is approximately 0.4 for total arsenic. For the log-probit extrapolation, the "probit" intercept A results from the following equation:

$$P(x) = \phi (A + \log_{10} [x])$$

where ϕ is the cumulative normal distribution function.

This equation makes the usual assumption that the log-probit dose/response curve has unit slope with respect to the log-dose. From tables of the standard normal distribution, A is found to be approximately equal to -.6 for total arsenic. These values were used to determine the probability of a response at various concentrations according to the above equation.

The multistage model with a quadratic hazard rate function,

$$h(x) = ax^2 + bx + c,$$

was fitted to the same data. The parameters a, b, and c, were estimated by a maximum likelihood method, aided by a computer program that performed a heuristic search for the best fit. The parameter b dominates for small values of dose x, and the parameter a dominates for large values.

In Table 7-8 the risk estimates obtained from these three models are summarized. The expected number of cancers per million exposed population is shown for a wide range of concentrations in drinking water. These estimates represent probable upper bounds on the true risk, due to the conservative assumptions that were used. The gap between the estimates is large in the low-dose region; thus, there is a substantial range of uncertainty concerning the actual carcinogenic effects of arsenic. However, present scientific methods do not permit a more accurate or definitive assessment of human risk.

7.1.6.4 CAG Cancer Risk Estimate

The Carcinogen Assessment Group (CAG) has derived a model similar to the linear model presented previously, also based on the Taiwanese epidemiological study (U.S. EPA 1980). The model estimates a lifetime probability of developing skin cancer equal to 0.004 (4,000 out of 10^6 population) resulting from life-time exposure to arsenic at a concentration of 10 µg/l in drinking water. Although some of the basic assumptions of the model are slightly different from those of the linear model, the final estimate is the same.

TABLE 7-8. ESTIMATED LIFETIME EXCESS CANCERS PER MILLION POPULATION EXPOSED VIA INGESTION TO TOTAL ARSENIC AT VARIOUS CONCENTRATIONS BASED ON THREE EXTRAPOLATION MODELS[a]

		Estimated No. Excess Lifetime Cancers (per million population exposed)[a]						
Extrapolation Model	Concentration in Drinking Water (mg/l) =	10^{-7}	10^{-6}	10^{-5}	10^{-4}	10^{-3}	10^{-2}	10^{-1}
Linear Model		-	0.4	4	40	400	4000	39,000
Log-Probit Model		-	-	-	2	160	4700	55,000
Multi-Stage Model		-	0.6	6	60	600	6000	63,000

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[a] The number of lifetime excess cancers represents an increase in number of cancers over the normal background incidence, assuming that an individual is continuously exposed to total arsenic at the indicated concentrations in drinking water consumed at a daily intake rate of 2 l/day over their lifetime. There is considerable variation in the estimated risk due to uncertainty in the epidemiology study on which these numbers are based and the application of hypothetical dose-response curves. In view of several conservative assumptions that were utilized, it is likely that these predictions overestimate the actual risk to humans.

7.1.6.5 Implications of Taiwan Study for the U.S. Population

To put the results of the Taiwan study in perspective for the U.S., it is worth briefly considering the incidence of skin cancer in the United States and also pointing out possible differences between the Taiwanese study group and the American populace.

The dose-response relationship of increased frequency of skin cancer among inhabitants of the southwest coast of Taiwan to the arsenic content of drinking water utilized above in the calculation of risk, has been fairly well documented (Tseng *et al*. 1968). The mechanism of arsenical carcinogenesis, however, is still quite unclear. Attempts to induce tumors in animals have been largely unsuccessful. Furthermore, in contradistinction to the Tseng study, a survey of the population in the Lane County, Oregon, an area in the U.S. where significant amounts of arsenic are present in drinking water, showed no evidence of increased cancer incidence (Morton *et al*. 1976).

The best quantitative data on the incidence of non-melanoma skin cancer among Caucasians in the United States is contained in a 6-month survey of four areas of the U.S. (Dallas-Fort Worth, Minneapolis-St. Paul, San Francisco-Oakland SMSAs and the entire state of Iowa) during 1971-1972 (Scotto *et al*. 1974). The survey area encompassed approximately 5% of total U.S. population in 1970. The incidence of non-melanoma skin cancer encompasses approximately one-half of the total of all other forms of cancer combined in the United States (see Table 7-9), with the projected incidence for the entire U.S. approaching 425,000 cases per year.

Several differences between the arsenical skin cancer observed in Taiwan and skin cancer found in the United States, however are worthy of note:

- In the U.S., 80% of all non-melanoma skin cancers occur on exposed body sites (e.g., face, head or neck) (Scotto *et al*. 1974). In Taiwan, 60% of skin cancers are located in unexposed parts of the body (e.g., legs, feet, toes, trunk) (Yeh 1973).
- The incidence of skin cancer in the U.S. appears to reflect geographic latitude, with greater risk to residents of lower latitudes (Scotto *et al*. 1977). Skin cancer incidence rates appear to double for about each ten degree decrease in latitude (Cutler *et al*. 1974).
- No significant increase in cancer was found in residents of the Lane County, Oregon, despite high arsenic levels in water supplies, although admittedly much lower on average than levels reported from Taiwan (Morton *et al*. 1976).

TABLE 7-9. INCIDENCE RATES AMONG CAUCASIANS FOR NON-MELANOMA SKIN CANCERS, MELANOMA OF SKIN, AND CANCERS OF ALL ORGANS COMBINED FOR FOUR AREAS OF THE UNITED STATES

	Cancer Incidence per 10^6 Population[a]		
Area	Non-Melanoma[b]	Melanoma	All Sites[c]
Dallas-Fort Worth SMSA	3790	72	2556
Iowa	1240	34	3247
Minneapolis-St. Paul	1510	33	2789
San Francisco-Oakland	1840	60	3543
Average	2095	49.8	3034

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[a] Standardized to population of United States for 1970.

[b] Incidence rate for non-melanoma skin cancer was age-adjusted; the other rates were crude rates, which were not adjusted for a relatively small difference in age distribution of study population, to that of the entire United States.

[c] Excluding carcinoma *in situ* and non-melanoma skin cancers.

Source: Scotto *et al*. (1974).

Other factors that may have played a role in the positive association between arsenic ingestion and skin cancer in the Taiwanese include ethnic and environmental factors, socioeconomic status, occupation, personal habits (e.g., smoking) and diet. Nutrition can affect conditions of repair, immune competence, cellular integrity and skin features such as the keratin layer.

The estimated potential carcinogenic risk for humans due to ingestion of arsenic (total) in drinking water were derived from extrapolations based upon numerous assumptions. These result in estimating an extremely high incidence of skin cancer from exposure to low levels of arsenic. In fact, based on current U.S. cancer rate data, the relatively high incidence of skin cancer in the U.S. is primarily attributable to exposure to U.V. radiation based on the relationship between latitude and cancer rates. Because of the strong role of this factor in the U.S. skin cancer incidence, extrapolations of risk to specific subpopulations based on arsenic exposure levels alone seemed neither appropriate nor realistic. If the estimates were based on the results of a standard laboratory animal, long-term feeding, dose-response study where the actual amount of arsenic intake was known, then such extrapolations could be better justified. In the case of the Taiwanese study, however, the arsenic intake is only estimated so that projections onto another subpopulation with different eating habits and exposure routes are precarious.

7.1.6.6 The Relationship Between Inhalation of Arsenic and Carcinogenicity

Respiratory cancer has been associated with exposure to sources which release arsenic (among other pollutants) to the air (Wagner 1973, U.S. EPA 1980). Evidence for this association is not thought to be as good as the evidence relating arsenic to skin cancer (Mushak *et al*. 1980). Other metals and SO_2 are also often released in these emissions so that interpretation of observed effects is complex and not entirely attributable to arsenic. Histological interpretation of the resulting carcinomas has been minimal. Also the role of arsenic as a co-carcinogen in the presence of other toxins is not well understood. As is true for other routes of exposure (ingestion), laboratory attempts to confirm arsenic's status as a respiratory tract carcinogen have been relatively unsuccessful.

The Carcinogen Assessment Group's (CAG 1980) quantitative risk assessment of the airborne route of exposure to arsenic calculated a lifetime risk of respiratory cancer of 1×10^{-5} for approximately 2.16×10^6 people for all air arsenic exposures. Populations in the vicinity of cotton gins had an estimated risk of cancer as high as 8.77×10^{-3}. The total respiratory cancer due to airborne exposure was estimated at 0.004% of the U.S. total. CAG estimated that the rate of developing lung cancer over a lifetime increased by approximately 8% per $\mu g/m^3$ increment increase in atmospheric arsenic concentration (Mushak *et al*. 1980).

7.1.7 Areas for Future Research

Research areas that require further investigation include: the role of arsenic as a factor in dermal and lung carcinogenicity; the possible role of co-factors in carcinogenicity; teratogenic and mutagenic effects of arsenic; and indirect effects of arsenic on other bodily functions, e.g., disease resistance. Data gaps in the literature make it difficult to resolve several issues with respect to the human health effects of arsenic. This is partially the result of complications due to the variability of the chemical forms.

7.1.8 Conclusions

The risk associated with exposure of humans to adverse levels of arsenic in the environment is uncertain. Based on extrapolations from a Taiwan epidemiology study showing a relationship between skin cancer and low drinking water concentrations of total arsenic, risk estimates of excess individual lifetime skin cancer incidence associated with ingestion of 3 μg/l of arsenic in drinking water range from 0 to a range of 0.0009 to 0.0018. Three micrograms per liter is a commonly occurring arsenic concentration in water supplies. The implications of this very high rate of skin cancer for the U.S. must be qualified by several points: 1) attempts to induce tumors in laboratory animals exposed to arsenic have been unsuccessful; 2) other potentially harmful chemicals were present in Taiwan ground water (lysergic acid,ergotamines) 3) the chemical form of arsenic in U.S. ground water may not be the same form as found in the Taiwanese study; 4) no significant increase in cancer was found in an epidemiology study of Lane County, Oregon in which relatively high arsenic levels are present in some of its water supply; 5) although the non-melanoma skin cancer rate is high in the U.S., the cancers tend to be located in different parts of the body than the Taiwanese cancers and there is a strong relationship between incidence rates and geographic latitude. Therefore, even without considering the inherent weak epidemiology studies themselves, there is a considerable amount of uncertainty associated with the extrapolation of the Taiwan skin cancer rate to the U.S. population.

Aside from the uncertainties inherent in the type of data used in the risk analysis, other important and largely unquantifiable sources of uncertainty exist. In that the mechanisms of carcinogenesis are not fully understood, there is no present scientific consensus for selecting the most appropriate model among a variety of risk extrapolation models. These models make similar risk estimations at high exposure levels, but markedly different estimations at the relatively low levels that are of concern with environmental exposures. In addition, the lack of control groups and contradictory results from equally valid studies (i.e, Lane County) add to the uncertainty.

The risk associated with exposure to adverse environmental levels is lower if one considers the effects reported for other chronic exposure to low levels of various forms of arsenic in laboratory studies. Peripheral neuropathy resulted from exposure to the lowest reported effects level of 0.04 mg/kg/day of calcium arsenate ingested for 2-3 weeks. Other effects resulting from exposure to arsenate were observed at concentrations of 10 mg/kg or higher; concentrations of 23 mg/kg/day to 93 mg/kg/day did not elicit carcinogenesis in rats. Data for arsenite were very limited; skin disorders resulted from exposure to 0.13 mg/kg/day and no evidence of carcinogenesis was found in rats exposed to arsenite at levels of 13 mg/kg/day. Data for arsenic (unspecified), in addition to the Taiwan study, indicated central nervous system damage and lethality in infants exposed to 0.8 mg/kg/day and 7.9 mg/kg/day, respectively. The U.S. Interim Drinking Water Standard for total arsenic is 50 µg/l, which is equivalent to an intake level of 0.001 mg/kg/day from drinking water. In general the data base for the toxicity of arsenic and its compounds is very poor.

Average exposure levels of arsenic for the general U.S. population through ingestion are less than 0.001 mg/kg for drinking water, total diet, and wine or moonshine. Consumption of contaminated fish or ground water may result in considerably higher levels exceeding 0.01 mg/kg/day. However, a low absorption efficiency of arsenic from ingestion of seafood has been reported. Dermal exposure for the majority of the U.S. population is expected to be negligible. Contact with arsenic-treated wood and contaminated detergent is associated with intake levels of 0.004 mg/kg/day and 0.06 mg/kg/day, respectively. However, the subpopulation exposed through these routes is small. Exposure of the general population through inhalation of ambient levels of arsenic is negligible and less than 0.0001 mg/kg/day in the vicinity of sources. Cigarette smokers may be exposed to >0.001 mg/kg day. Therefore the highest exposure levels for the general population are from ingestion routes.

Distinguishing between chemical forms of arsenic associated with different exposure routes is complicated due to a general lack of data, poor analytical techniques, and the presence of multiple forms in most media. However, some exposure media with potentially high arsenite levels include well water, wine and some fish tissue. Due to the uncertainty of these data, risk is estimated for exposure to total arsenic levels. Therefore different exposures levels can be combined into exposure scenarios for selected subpopulations to better represent total exposure to arsenic.

The total estimated exposure to arsenic for the general non-smoking population and for children is approximately 0.4 µg/kg/day and 8 µg/kg/day, respectively. Smokers may be exposed to 2 µg/kg/day. Subpopulations with diets including ground water, fish or wine highly contaminated with arsenic may be exposed to from 7 µg/kg/day to 143 µg/kg/day. Woodworkers may be exposed to 4 µg/kg/day to 7 µg/kg/day. The worst case, assuming exposure to high but not the maximum levels of arsenic in all media, has an associated exposure of 41 µg/kg/day.

The risk of human exposure to adverse levels of arsenic is low for the general population considering that the total intake level of 0.4 µg/kg/day is lower than the lowest available reported effect level for any arsenic compound of 40 µg/kg/day (peripheral neuropathy). In fact, most subpopulations have exposure levels lower than the acceptable intake level suggested by the U.S. Interim Drinking Water Standard. Subpopulations potentially at risk (with levels exceeding lowest effects levels) include people with large amounts of contaminated fish or ground water in their diets. The sizes of these subpopulations are unknown at this time due to limited information regarding the national distribution of ground water containing arsenic and of fish contamination.

A very limited amount of data are available on the toxicity of arsenic compounds to humans and laboratory animals. In addition, the available data do not always distinguish between chemical forms of arsenic nor does it adequately cover all the forms. The Taiwan study indicates adverse effects resulting from lower concentrations than toxic effects levels reported in other studies. Therefore, any conclusions made about the risk of humans in adverse exposure to arsenic are tentative and subject to change with additional data.

7.2 RISK CONSIDERATIONS FOR NONHUMAN BIOTA

7.2.1 Statement of Risk

Arsenic in surface water does not appear to present a significant risk to aquatic organisms with respect to acute adverse effects. The levels (usually as dissolved As), which are reported to be toxic to a large group of aquatic species are usually two to three orders of magnitude greater than the range of ambient levels in surface water most commonly observed in the U.S. (reported as total As). Other substances present in environmental waters such as suspended organic matter or hydrous oxides would reduce even lower the fraction of total arsenic actually available for biological uptake, making the margin between effects and ambient levels even wider. Soil levels of arsenic high enough to have adverse effects on plants and microbial communities are likely to accumulate in the vicinity of certain industrial sources but do not appear to be widespread on a national basis.

7.2.2 Background

Chapter 6.0 described the effects levels of dissolved arsenic found to have sublethal and lethal effects on fresh and saltwater aquatic species (6.1) and the levels (total arsenic) in the environment to which these species are likely to be exposed (6.2).

The majority of mean ambient levels of arsenic in U.S. surface water by major river basin over the past 5 years are less than 0.01 mg/l; 9% are between 0.01 mg/l and 1.0 mg/l and less than 1% are greater than 1.0 mg/l. Table 7-10 summarizes ranges in arsenic levels that elicited effects in aquatic organisms under laboratory conditions. The ranges are grouped by chemical form. Figure 7-1 provides a graphical representation of the same data for total arsenic. Even considering the most toxic form, the trivalent (sodium arsenite), the effects levels reported for the most sensitive species for which data were available, immature bluegill and Daphnia--are only rarely found in U.S. waters.

7.2.3 Local Regions of Potential Risk

Two areas of the U.S. were focused on through examination of a minor river basin data: the Tacoma, Washington area and the Missouri River Basin (see Chapter 6.2). The Tacoma site was chosen because it is the location of the only U.S. arsenic producer. Although the prouction plant currently has negligible aquatic releases, the influence of past discharges and releases to air and soil on water concentrations was considered. The Missouri area was chosen for consideration due to the occurrence of high mean water concentrations in the major river basin over the past 10 years. The Tacoma site indicated no concentrations of total arsenic at levels eliciting effects in even the most sensitive species as determined in laboratory bioassays. Mean sediment levels in the river basin were no greater than levels in other parts of the U.S. which suggested that arsenic was not accumulating in the sediment. These conclusions are based on a limited set of data, however, and therefore may be invalidated by additional data.

Data for the Missouri Major River basin revealed high levels of arsenic, in terms of aquatic effects, in two minor river basins located in South Dakota and Montana. In one minor river basin, concentrations reported as recently as 1979 also exceeded the effects level for a number of freshwater species. The presence of these levels is possibly due to the presence of large fossil fuel combustion plants, weathering and leaching of continental rock, low stream flow rates at the time of monitoring, or other factors. The data indicate the likelihood of a toxicity problem for aquatic life in this region of the United States as indicated by total arsenic levels. To better understand the actual impact of these concentrations on aquatic communities at this location, concentrations of available, soluble arsenic are necessary. Also, some qualitative evaluations of the availability could be made if the concentrations of complexing agents and adsorbing material were known for that location.

TABLE 7-10. RANGES IN EFFECTS LEVELS REPORTED FOR AQUATIC SPECIES GROUPED BY ARSENIC FORMS

Arsenic Species/ Concentration Range (mg/l)	Effects
Sodium Arsenite	
0.1 - 1.0	Acutely toxic to bluegill fingerlings, and to cladoceran.
1.0 - 10.0	Range of toxic effects to freshwater invertebrates and to marine invertebrates including scallops and oysters; threshold of toxic effects such as reduction in numbers in bluegill.
10.0 - 50.0	Range of toxic effects to most freshwater fish and to stoneflys.
Sodium Arsenate	
1.0 - 10.0	Acute effects to most sensitive species, Daphnia magna.
10 - 200	Acute effects to other species (green sunfish, Lepomis cyancellus).
Arsenic Trioxide	
1.0 - 10.0	Metabolic effects on rainbow trout; reduced survival in several freshwater invertebrates, including shrimp, amphipods and insects, acutely toxic to chum salmon.
Arsenic Trisulfide	
25	Toxic to white shrimp (Penaeus spp.)
>25 - 700	LC_{50} to fathead minnow and freshwater insects.

Source: Section 6.1.

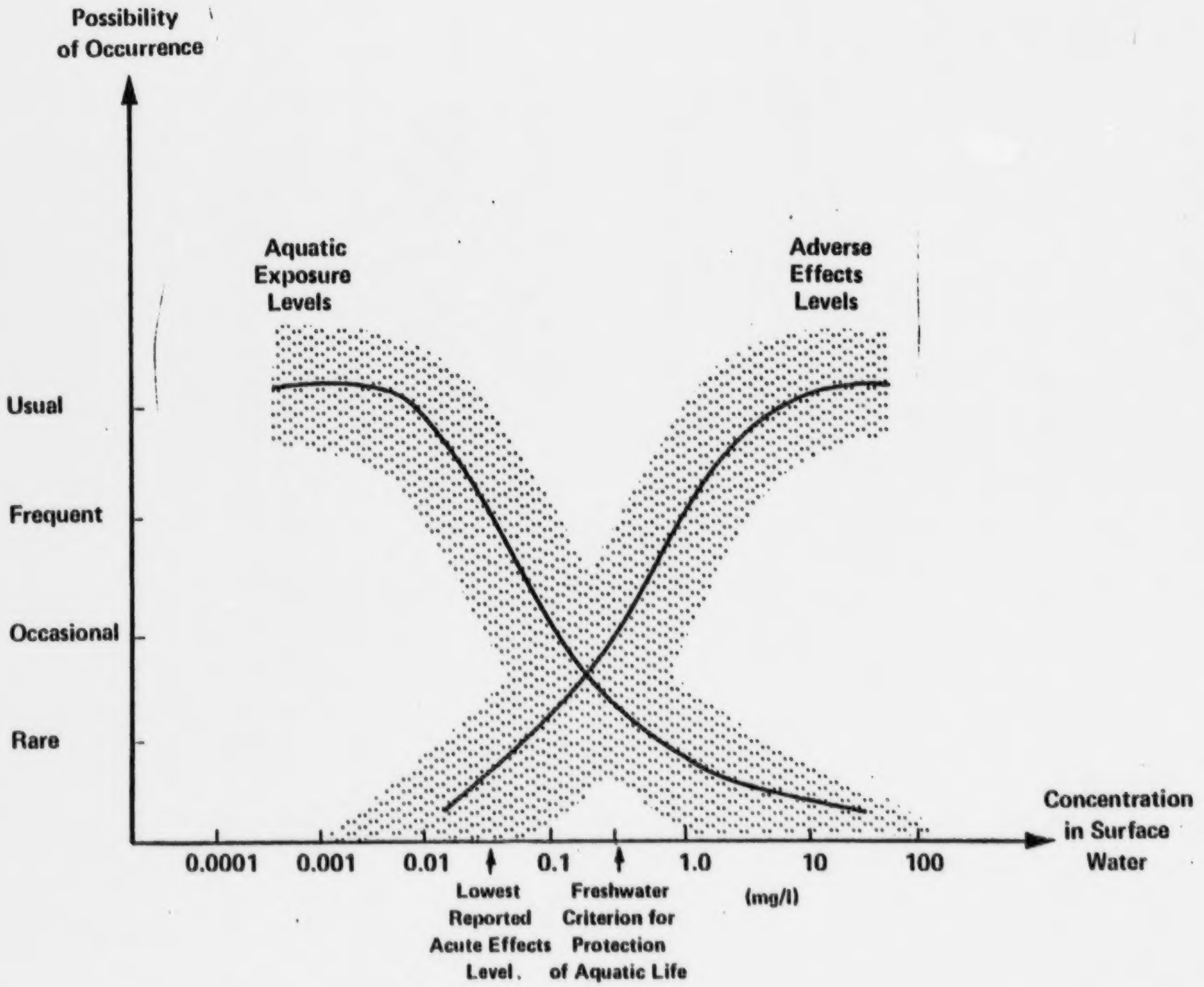

FIGURE 7–1 COMPARISON OF AQUATIC EXPOSURE AND EFFECTS LEVELS – TOTAL ARSENIC

Other major river basins in which high mean levels (relative to the national mean) were observed included the Great Basin, Lower Mississippi and Western Gulf basins. It is possible that the slightly higher levels were due to higher overall industrial activity in these locations and/or the use of arsenic pesticides in the South. A more detailed level of analysis, however, would be required of the minor river basins and individual stations comprising these basins in order to address these uncertainties.

7.2.4 Aquatic Herbicide Use

Another source of high arsenic levels in surface water, with possible adverse effects on aquatic systems, is the use of sodium arsenite as a herbicide for aquatic vegetation. Application levels may be as high as 10 mg/l ($\approx$ 7.6 mg/l As). Equivalent concentrations have resulted in toxicity in laboratory bioassays. However, in a lake in Wisconsin there was no observable effect at these levels on fish populations; no fish species were specified (Lambou and Lim 1970). The effective concentration of arsenic and duration of this level in a natural system will depend upon the rate of application, the redox environment, the sensitivity and previous history of species present, the concentration of adsorbing material (e.g., ferrous oxides, organic matter, etc.), as well as other factors. The sediment may provide a reservoir for a large amount of the arsenic applied (see Section 4.2).

No information was available on the predominance of sodium arsenite use as an aquatic herbicide nor is a sufficient amount of data available on field observations of effects to analyze the impact of such uses. The total number of aquatic systems potentially affected is expected to be small, however, and confined to warmer climates where herbicides are consistently used to control aquatic vegetation overgrowth (e.g., water hyacinths).

7.2.5 Sensitive Species

The freshwater fish species reported most sensitive to acute exposure to arsenic (sodium arsenite) was the bluegill (Lepomis macrochirus) at the fingerling stage, with an LC_{50} of 0.29 mg/l. Adult bluegills, however, were much more resistant to arsenic. A total of eight species were tested; thus it is possible that more sensitive species do exist but were not included in the available studies. The most sensitive freshwater invertebrate out of the four species tested was the cladoceran, Simocephalus serrulatus, with an LC50 of 0.8 mg/l. Both of the sensitive species have a widespread distribution within the continental U.S. As discussed previously, the occurrence of acute arsenic levels sufficient to cause adverse effects in even these most sensitive species is rare on a national basis.

7.2.6 Terrestrial Ecosystems

Considering the large amount of arsenic deposited onto land in solid wastes and pesticide application, there would at least appear to be a high potential for exposure and risk of terrestrial plant and microbial communities. The potential for mixing and movement of arsenic in soil (excluding migration in aquifers) is much lower than in water or air; therefore, the effects of high concentrations of the pollutant in one spot are usually localized. This makes it possible to identify problem sites and avoid cultivation of food or cash crops where contamination or yield reductions could occur. Areas of potential risk are spotty and relatively contained, and hence arsenic is unlikely to spread into areas important in terms of human or wildlife exposure. This does not, however, apply to arsenic that has migrated vertically through the soil into ground water.

In the vicinity of certain areas, either in soils with high natural background levels of arsenic or contaminated by human activities, arsenic concentrations are high enough to reduce plant growth or lead to significant bioaccumulation. Natural levels of greater than 100 mg/kg have been reported in soils overlying sulfide ore deposits (NAS 1977). As described in Chapter 6.2, soil that has received pesticide applications (i.e., orchards, blueberry and potato fields), industrial wastes (i.e., flyash, municipal wastes, slag) or lying downwind from smelting operations can accumulate arsenic levels from 150 mg/kg to 25,000 mg/kg (total As). Comparing these exposure concentrations to the effects levels for crops reported in Chapter 6.1 of 25 mg/kg to 85 mg/kg, it is evident that growth of the species in which these effects were measured, and probably most other plants, would be severely reduced in the situations described previously.

Most plant species are so sensitive to arsenic that it is thought that tissue levels high enough to harm humans and wildlife ingesting contaminated food are unlikely to accumulate in the edible fraction before the plant itself is subject to growth reduction (Woolson 1969). Exceptions to this observation may include root crops, especially potatoes that accumulate high concentrations in the peel, leafy plants, and perhaps plants treated with organoarsenicals, which are more effectively translocated than the inorganic forms into the edible part (Walsh and Keeney 1974). Organoarsenicals are applied to soil at lower rates than are inorganic forms, however, and this would subsequently reduce uptake. The monitoring data indicate generally low levels of arsenic of all forms in fruits and vegetables, even root crops. The exposure of humans to contaminated food crops is discussed in Chapter 5.2.

It is more difficult to estimate the potential risk of microbial populations than for plants living in soil with high arsenic levels. This is due to the limited information available regarding inhibitory levels for microbial activity in other than simplified laboratory systems. In many of the arsenic-contamination situations described above, however, the concentrations are probably high enough to inhibit organic matter turnover rates, nitrogen and other element cycles, and the cycling of arsenic itself. These situations are limited and localized enough so that the impact would not be great on an ecosystem level. The impact of lower levels of arsenic, which are more widespread, or of the various chemical forms of arsenic, is unknown.

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APPENDIX A

Note 1

Atmospheric emission estimates are based on EPA 1979a and 1979b. Uncontrolled fugitive emissions of arsenic from the ASARCO, Tacoma smelter are calculated from the following emission estimates and 365 day per year operation, 16 hours per day.

Fugitive Emissions		As kkg/yr
Calcine transfer from roaster	11.2 lb/hr x 365 d/yr x 16hr/day	30
Transfer, flue dust, handling	3.8 lb/hr	10
Arsenic building	1.8 lb/hr	5
	total fugitive emissions	45
Controlled Emissions		
From stack	62.4 lb/hr x 365 d/yr x 16hr/day	166
	total emissions	210

Arsenic is associated with other emissions at the Tacoma smelter; these are included in inadvertent sources - copper production since they are not directly involved with As_2O_3 recovery.

Note 2

Based on EPA 1979a/b and EPA, 1975. Slag at ASARCO, Tacoma is dumped and air cooled instead of granulated. It is assumed to be disposed to land, although it can be used as road construction material. Land-destined wastes are calculated as follows:

Reverberatory furnace slag: 450 lb As/hr x 365 d/yr x 16 hr/day = 1195 metric tons/yr. Arsenic-containing solid wastes are generated at other locations in the ASARCO, Tacoma facility. These are included in inadvertent sources - copper production as they are not directly a part of As_2O_3 recovery.

Note 3

Based on total pressed and blown glass manufacture of 2,993,100 kkg per year (EPA, 1980b). About 77% of that (2,304,690 kkg) is soda lime glass which presently does not use arsenic as an additive (EPA, 1980b; Thatcher Glass Co., 1980). The remaining 688,410 kkg consists of 11% borosilicate glass, 5% lead glass, and 7% opal glass (EPA, 1980b). If 25% of the borosilicate production involves arsenic, as well as all of the lead and opal glass, 634,900 kkg of pressed and blown glass produced annually contains arsenic (EPA, 1980b). Based on a controlled emission factor of 0.015 kg As emitted per kkg of pressed and blown glass produced, 10 kkg of As are emitted annually from glass manufacture.

Note 4

Less than 1 kkg of arsenic is discharged to water (chiefly POTWs) from wood preserving plants annually. Based on raw waste concentrations and flow rates from plants using organic preservatives only and a total of 476 plants (AWPA, 1979) approximately 0.2 kkg of arsenic would be discharged (Table A-1). This figure represents a maximum since the number of facilities using organic preservatives only was not available.

Plant average arsenic loading kkg/yr x total # of plants = As discharge
0.0004 kkg/yr/plant x 476 plants = 0.2 kkg

Alternatively, using the average discharge for plants treating with organic and inorganic preservatives, the annual discharge of arsenic would be 0.5 kkg per year (Table A-2). Both of these estimates are raw waste concentrations; wastewater treatment would lower these discharges. Most plants are already achieving zero discharge.

Note 5

A. 92,903,000 ft^3 wood treated x 0.016 yd^3 wet sludge/1000 ft^3 wood treated = 1,486 yd^3 wet sludge/yr.

B. $\frac{.765\ m^3}{yd^3}$ = 1,486 yd^3 = 1,137 m^3 wet sludge

C. Density = $\frac{mass}{volume}$ 1.03 x 1,137 = 1, 171 kg wet sludge

D. If wet sludge is 95% water, then this sludge would yield 58 kg solids (dry sludge).

E. 9 gm As/kkg dry sludge x 0.058 kkg dry sludge = 0.5 g As or $5x10^{-4}$ kg As/yr.

Note 6

No data were found concerning wastewater discharges from individual smelters; EPA, 1975a addresses the primary copper industry as a combination of smelters and refiners. Therefore a total arsenic discharge for the entire primary copper industry was obtained by calculating an average As discharge per plant and applying that average to the total number of facilities. Assume 350 d/yr operation.

Plant	Effluent Loading (kg/kkg production)	Production (kkg/day)	As discharge kkg/yr
115	0.026	494	4.4
116	0.0003	415	0.04
117	0.174	293	17.8
118	0.0007	454	0.1
121	0.0002	263	0.02
102	0.003	311	0.33
110	0.0001	674	0.02
		average	3.2

Currently, there are 16 copper smelters and 11 refineries in the U.S. Four of the refineries are located on the same site as the smelter. Assuming that these 4 refineries combine their wastewater with their respective smelters, and discounting the ASARCO, Tacoma facility which is included in arsenic production, a total of 19 plants would be discharging 3 kkg As/yr or a total industry discharge of 60 kkg As/yr. However, most smelters and refiners are at or approaching zero discharge. Based on 50% of plants applying treatment and/or recycling wastewater, 30 kkg As would be discharged per year. This discharge is usually sent to tailing ponds EPA, 1975a.

Note 7

Based on effluent loadings of 8 secondary copper facilities and applied to the 50 total plants. Assume 350 day/yr operation.

Plant	Effluent Loading (kg/kkg production)	Production (kkg/day)	As discharge kkg/yr
1	0.0009	529	0.17
8	----*	327	0.01
9	----*	47	0.001
12	0.0001	78	0.003
26	-0-	33	-0-
32	-0-	150	-0-
39	0.000001	44	0.00002
43	----*	62	0.002
		average	0.02

*For effluent loadings not listed, 0.0001 was used.
50 plants x 0.02 kkg As/yr plant = 1 kkg As discharged

Note 8

EPA, 1979b. Based on raw wastewater As concentrations and flow rates for 4 primary lead smelters. The average was applied to the total plants. Assume 350 day/yr operation.

Plant	As mg/l	Flow l/day x 350 d/yr	As kkg/yr
A	.130	2.4×10^6	0.1
B	.018	4.5×10^6	0.03
C	.093	8.3×10^6	0.3
D	0.10	4.9×10^6	0.02
		average	0.11

7 plants x 0.11 kkg As/yr/plant = 0.78 kkg As

Note 9

EPA, 1979b. Based on treated wastewater As concentration and flow rates for secondary lead smelters. The average per plant was applied to the total of 69 plants. Assume 350 day/year operation.

Plant	As μg/l	Flow l/day	As kkg/yr
A	2.9×10^{3}	0.015×10^{6}	1.5×10^{1}
C	10	0.01×10^{6}	3.5×10^{-5}
D	25	0.11×10^{6}	9.6×10^{-4}
		geometic mean	8.0×10^{-3}

69 plants x 8.0×10^{-3} kkg As/plant/yr = 0.6 kkg yr

Note 10

EPA, 1978a, and Plunkert, 1980. Antimony is produced by either pyrometallurgic or electrolytic processes, each of which generate a specific type of arsenic-containing waste. If half of the total 2,400 kkg of metallic antimony is produced by each method, then:

Pyrometallurgic:

$$\frac{2{,}800 \text{ kg slag}}{\text{kkg antimony produced}} \times 1{,}200 \text{ kkg antimony produced} \times \frac{0.003 \text{ kg As in slag}}{\text{kkg slag}} = 10 \text{ kkg As}$$

Electrolytic:

$$\frac{210 \text{ kg solids (in sludge)}}{\text{kkg antimony}} \times 1{,}200 \text{ kkg antimony} \times \frac{0.016 \text{ kg As}}{\text{kkg}} = 4 \text{ kkg As}$$

Therefore, a total of 14 kkg As of land-destined wastes are generated from antimony manufacture.

Note 11

A. Figures reported are for primary zinc processing only since all secondary plants are achieving zero discharge (EPA, 1979b). Of the 6 zinc processing facilities, 5 are direct dischargers, 1 is indirect. An average treated wastewater discharge per plant was calculated from As concentrations and flow rates from 3 plants. Assume 350 day/yr operation.

Plant	Flow (10^6 l/day)	As mg/l	As kkg/yr	
B	2.15	0.92	0.7	0.7
B	1.59	0.002	0.001	
C	1.63	4.8	2.7	
A	0.09	0.001	0.00003	0.00063
A	1.81	0.001	0.0006	
		average	1.1	

5 direct dischargers x 1.1 kkg As/yr/plant = 6 kkg
1 indirect discharger x 1.1 kkg As/yr/plant = 1 kkg

B. Of the 30 primary aluminum processing facilities, 27 are direct dischargers, 3 are at zero discharge (EPA, 1979b). Only 3 of the 27 direct dischargers provide no treatment. An average discharge per plant was calculated from As concentration in treated wastewater, and flow rates for 5 plants. Assume 350 day/yr operation.

Plant	Flow (10^6 l/day)	As mg/l	As kkg/yr	
A	60.6	0.01	0.2	
B	1.4	0.05	0.02	0.024
B	1.2	0.01	0.004	
D	6.0	0.035	0.07	
C	21.9	0.01	0.08	
E	0.32	0.001	0.0001	0.24
E	36.3	0.019	0.24	
E	0.07	0.007	0.0002	
		average	0.12	

27 direct dischargers x 0.12 kkg As/plant/yr = 3 kkg As

Note 12

Of the 55 domestic copper mines, 10 discharge mine water to surface waters (based on EPA, 1975c survey of 21 mines, 4 of which discharged mine waters) the remainder recycle/reuse such water in milling processes (EPA, 1975c). Half of the dischargers treat the water prior to discharge. Assuming treatment efficiency of 50% and 350 day/yr operation, 6 kkg As/year are discharged from copper mines, based on As concentrations and flow rates for 2 mines (EPA, 1975c).

Mine	Flow (10^6 l/day)	As mg/l		As kkg/yr
2119	42.0	0.07		1.0
2120	27.5	0.07		0.7
			average	0.85

.50 efficient x 0.85 kkg As/yr/mine x 5 mines = 2 kkg As
0.85 As/yr/mine x 5 mines (no treatment) = 4 kkg As

Copper mill wastewater is discharged to tailing ponds for settling; water is then recycled within the plant. Based on raw waste loadings for tailing pond influent, and a total production of 62,180,500 kkg in 1979 (Butterman, 1980) approximately 640 kkg of As are discharged to tailing ponds per year (EPA, 1975c).

Mill	raw waste loading kg As/1000 kg Cu produced	annual concentrate production kkg	discharge kkg As/yr
2119	11.77	454,420	5
2120	0		0
2121	7.33	223,318	2
2122	9.38	740,602	7
2123	25.97	11,170	0.3
2124	7.06	69,362	0.6
Average	10.3		2.5

10.3 kg As/1000 kkg Cu produced x 62180.5 x 10^3 kkg Cu produced = 640 kkg As.

Only three major mills discharge tailing pond water to surface water. Based on an average discharge of 2.5 kkg As/mill, 8 kkg of As are discharged per year. Therefore, about 630 kkg As remain in tailing ponds and presumably discharged to land.

Note 13

EPA, 1976a. As little data are available concerning arsenic in the smelting of manganese, it is assumed that arsenic behaves similarly to phosphorus: 60% remains in ferroalloy, 30% contained in slag, 10% emitted to the atmosphere. Therefore an annual consumption of 2 x 10^6 kkg manganese, 90% of which is used in ferroalloy production, containing 0.2% As yields 2,160 kkg remaining in ferroalloy and 1,080 kkg sent to land as slag. A 97% collection efficiency results in 360 kkg sent to land from collected flue dust and 10 kkg emitted to the atmosphere. The remaining 400 kkg are assumed to be contained in carbon zinc batteries and manganese-containing chemicals.

Note 14

EPA, 1976a. Residue from digested borax ore totals 800 kg/kkg borax products and contains 45 ppm As. Assuming 860,000 kkg borax products produced in 1976 is similar to 1979 production, approximately 30 kg of As are contained in wastes/year. These wastes are normally sent to ponds.

Based on 12 x 10^9 liters/yr of brine processed and an arsenic concentration of 180 mg/l, approximately 2,200 kkg of arsenic are contained in brine (EPA, 1976a). Depleted brines are returned to the lake from which they were extracted. Since arsenic is not intentionally removed from brine, it is assumed that all arsenic present is returned to the lake.

Based on an annual production of 110,000 kkg, 4 kkg of arsenic is discharged per year from boric acid production. The specific aquatic sink, surface water or POTW, for this discharge is unknown.

Note 15

Based on emission estimates from Davis and Associates, 1971, and Bureau of Census, 1979. Particulate emissions from cotton gins average 5.3 kg/bale cotton; average arsenic concentration of particulates in 300 mg/kg. Therefore 5.3 kg particulate/bale x 10,549,219 bales x 300 mg As/kg particulate = 17 kkg As. Trash from ginning operations is either burned (37%), disposed on land (58%) or handled in an unspecified manner. About 76% of the arsenic in trash (assumed to be 300 mg/kg as is in particulates) is emitted to the atmosphere. Therefore:

1,237,318 kkg trash burned x 300 g/kkg x 76% emitted = 281 kkg As.
1,939,579 kkg trash disposed to land x 300g As/kkg = 581 kkg As.
Total As to air = 298 kkg
As to land = 581 kkg

Note 16

Arsenic is added to lead shot, bearings and batteries. Refining temperatures (260-270°C) are low compared to vaporization temperature of arsenic (615°C) EPA, 1980b. EPA, 1976a estimates 0.75 kkg As is released from all nonferrous alloy production.

Note 17

EPA, 1976a. Of the total rock produced, 49% is used as domestic fertilizer, 4% as animal feed, 5% used for detergents, 3% for food products, 3% miscellaneous; the remainder is exported. Quantities of arsenic contained in each category are given in Table A-3. Arsenic is intentionally removed from food additives and phosphorus oxychloride. It is estimated that all of the arsenic thus removed is disposed to land (EPA, 1976a) as well as 50% of the arsenic contained is miscellaneous uses; total land disposal As = 130 kkg As. That arsenic contained in fertilizer (460 kkg) and animal feed (50 kkg) is also land disposed; most arsenic contained in detergents (160 kkg) is assumed released to water.

Note 18

Based on EPA, 1976a; EPA, 1979e; Desy, 1978. Pig iron production in 1977 totalled 7.37 x 10^7 kkg (Desy, 1978); average As concentration is 400 mg/kg (EPA, 1979e). Therefore 7.37 x 10^{10} kg x 400 mg/kg = 2.95 x 10^4 kkg As or 29,500 kkg As contained in pig iron. Almost 94% of pig iron (6.9 x 10^7 kkg) is consumed in steelmaking (EPA, 1976a), the remainder (4.4 x 10^6 kkg) is used in cast iron. Additionally, about 15 x 10^6 kkg of scrap is used in cast iron production. No arsenic concentration was found, therefore it is assumed to be similar to that of pig iron (650 ppm). Using an uncontrolled emission factor of 0.007 kg As/kkg metal, about 136 kkg of arsenic would be emitted from the 19.4 x 10^6 kkg cast iron production. Control efficiencies are assumed to be 95%. Therefore, approximately 129 kkg of arsenic are caught in pollution control devices and eventually landfilled; the 7 remaining kkg are emitted to the atmosphere.

Based on Copeland, 1980 nationwide estimates of particulates emitted from open hearth, electric arc, and basic oxygen steel furnaces are 27,570 kkg, 21,770 kkg, and 19,500 kkg respectively. If the arsenic content of particulates is similar to that of the pig iron being charged (400 mg/kg) then arsenic emissions from steel manufacture are as follows:

Open hearth - 27,570,000 kg x 400 mg/kg = 11 kkg
Electric arc - 21,770,000 kg x 400 mg/kg = 9 kkg As
Basic oxygen - 19,500,000 kg x 400 mg/kg = 8 kkg As
Totals 68,840,000 kg particulate 28 kkg

Steel making dusts are assumed to be landfilled. Assuming collection efficiencies of 95%, the emitted particulates should be 5% of the total. By difference 5,510 kkg As are landfilled as steelmaking dusts:

68840 = 0.05 y
y = 1,376,880 kkg particulates trapped
x 400g/kkg = 5510 kkg As landfilled

Arsenic contained in steel making slag is estimated to be 34,600 kkg per year. Most of the slag is used as railroad ballast, aggregate for concrete or recycled within the plant; the arsenic is thus considered to be contained and not released to the environment. Additionally, about 14,600 kkg of arsenic are estimated to be contained in steel production (EPA, 1976a).

Wastewaters from iron and steel plants stem from coke ovens, acid pickling, and casting, machining and hot and cold working of steel products. Based on EPA, 1979f data, ammonia liquor waste streams and cooler blow down contribute 42 kkg As per year in raw wastewater, assuming plants operate at 80% of capacity.

Operation	mg As/l	flow (l/ton)	total industry production kkg	kkg As
cooler blow down	0.005	118	3,430,630	0.002
NH_3 liquor	85.3	144		42
			total	42

Of that 42 kkg As, 14 kkg are directly discharged, 11 kkg are discharged to POTW's, 17 are recycled with quench water. Since quenching water is eventually evaporated, the As contained in it is assumed to be entirely emitted. These figures represent a worst case scenario as they are raw wastewater. Assuming 50% treatment application and efficiency for discharges, 7 kkg would be directly discharged, 6 are sent to POTWs and the 12 kkg collected would be sent to land.

Note 19

Based on 380,950 kkg secondary copper produced (Schroeder and Coakley, 1978), arsenic content of 5 mg/kg refined copper, (EPA, 1979e) and 350 kg slag generated/kkg secondary copper produced (EPA, 1978a).
380,950 kkg 2°Cu x 350 kg slag/kkg 2° Cu x 5 mg As/kg Cu x 1 kkg As

Note 20

Natural loading estiamtes based on the following data (as cited in EPA 1980e):

Worldwide quantity of material eroded: 9.3 x 10^9 kkg/yr
Portion of that total attributable to the U.S.: 6.3% or 5.86 x 10^8 kkg/yr

Eroded material contains 20% igneous rock (1.8 mg/kg As), 65% shale (6.6 mg/kg As) and 15% limestone/sandstone (1.5 mg/kg As)

Therefore:

0.20 x 5.86 x 10^{11} kg/yr x 1.8 mg/kg = 210 kkg/yr
0.65 x 5.86 x 10^{11} kg/yr x 6.6 mg/kg = 2,510 kkg/yr
0.15 x 5.86 x 10^{11} kg/yr x 1.5 mg/kg = 130 kkg/yr
2,850 kkg/yr

Approximately 2,850 kkg of As are released to the environment per year via weathering."Intensive" land use result in releases 4 times larger, or 11,400 kkg/yr.

Averaging these estimates, approximately 7,100 kkg/yr of As are released to the waters from erosion.

Note 21

Based on the following As concentrations (ppm) measured in urban runoff in various U.S. cities (EPA 1979j):

SanJose CA	<0.01
Orlando FL	0.04
New Creek, NY	31-14
Seattle, WA	0.049-0.060 $\overline{x}$ = 0.050
	0.05-0.1 $\overline{x}$ = 0.05
	0.05
	0.04-0.1 $\overline{x}$ = 0.05
	0.045-0.070 $\overline{x}$ = 0.05
	0.049-0.060 $\overline{x}$ = 0.05
	0.050

Table A-1. Raw Waste Arsenic Concentrations, Flow Rates, and Loadings for Wood Preserving Plants Using Organic Preservatives

Plant #	Flow (liters/yr)[a]	As (mg/l)	kkg As/yr
67	10,361,437	0.007	0.00007
267	12,081,720	0.093[b]	0.001
548	10,862,950	0.009	0.0001
591	4,636,625	0.086	0.0004
1,100	82,796,875	0.006	0.0005
		average	0.0004

a) Based on 350 day/year operation.

b) Higher value listed used in calculation to achieve maximum loading.

Source: EPA, 1979d.

Table A-2. Raw Waste Loading for Wood Preserving Plants Using Organic and Inorganic Preservatives

Plant #	Flow (liters/yr)	As (mg/l)		kkg As/yr
65	2,914,450	0.014		0.00004
237	2,649,580	0.050		0.0001
335	2,252,075	0.250		0.0006
499	132,475	1.00		0.0001
582	18,215,312	0.040		0.0007
897	56,169,400	0.130		0.007
1,078	20,268,675	0.003		0.00006
			average	0.001

a) Based on 350 day/year operation.

Source: EPA, 1979d.

Table A-3. Arsenic Releases from Primary Copper Smelting, 1979 (kkg/yr)

Source	Production		Environmental Releases				
	Input	Contained	Air	Land	Surface Water	POTW	Total
ASARCO							
El Paso	480[a]	240[b]	40[c]	220[d]			260
Hayden	400[e]	80[f]	210[g]	120[h]			330
KENNICOTT							
Hayden	64[i]	neg[j]	36[k]	40[l]			76
Hurley	2[m]	neg[n]	5[o]	neg			5
McGill	64[p]	2[q]	58[r]	8[s]			64
Garfield	1,100[t]	1,000[u]	8[v]	80[w]			88
PHELPS DODGE							
Douglas	80[x]	2[y]	37[z]	48[aa]			85
Morenci	80[bb]	3[cc]	30[dd]	48[ee]			78
Hidalgo	160[ff]	7[gg]	6[hh]	150[ii]			156
Ajo	540[jj]	31[kk]	310[ll]	220[mm]			530
Magma	32[nn]	neg[oo]	25[pp]	11[qq]			36
Copper Range/White Pine	6[rr]	neg[ss]	4[tt]	2[uu]			6 60
Inspiration	61[vv]	3[ww]	6[vv]	54[xx]			
Cities Service/ Copperhill	neg[yy]	_[zz]	_[zz]	_[zz]			0
Anaconda	6,700[aaa]	1,600[bbb]	180[ccc]	5,100[ddd]			5,300
TOTAL	9,800	3,000	960	6,100	30[eee]		7,100

Footnotes next page.

Table A-3. (Continued)

a) Based on .06 kkg As fed/hr (EPA, 1979a), 8000 hr/year operation (EPA, 1979a). All air and land releases from EPA, 1979a and are based on 8000 hr/yr operation. Totals may not add due to rounding.

b) Based on 0.03 kkg As in blister copper and dust returned to lead smelter.

c) Based on 0.004 kkg As to stack/hr, and 0.9 kg As/hr fugitive emissions.

d) Includes slag that is dumped and acid plant sludge. Based on 0.027 kkg As/hr.

e) Based on 0.05 kkg As/hr input.

f) Based on 0.01 kkg As in blister copper and material sent to El Paso/hr.

g) Based on 0.023 kkg As to stack/hr, and fugitive emission of 3 kg As/hr.

h) Based on 0.015 kkg As to dump in slag and as acid plant sludge/hr.

i) Based on 0.008 kkg As input/hr.

j) Based on 0.045 kg As/hr in blister copper. Negligible is defined as <1 kkg.

k) Based on 0.0035 kkg As to stack/hr, and fugitive emission of 1 kg As/hr.

l) Based on 0.005 kkg As in slag and acid plant sludge/hr.

m) Based on 0.0002 kkg As input/hr.

n) Based on 4.5×10^{-6} kkg As leaving in blister copper/hr.

o) Based on 0.13 kg As to stack/hr and fugitive emissions of 0.5 kg As/hr.

p) Input 0.008 kkg As/hr.

q) Based on 0.0002 kkg As/hr leaving in blister copper.

r) Based on 0.007 kkg As to stack/hr, and fugitive emissions of 0.3 kg As/hr.

Table A-3. (Continued)

s) Based on 0.001 kkg As to dump as slag.

t) Based on 0.14 kkg As input/hr.

u) Based on 0.13 kkg As leaving in blister copper and that sent to storage/hr.

v) Zero emissions from stack EPA, 1979a, fugitive emission of 1 kg As/hr.

w) Based on 0.01 kkg As/hr sent to dump in slag and in acid plant waste.

x) Input of 0.01 kkg As/hr.

y) Based on 0.0002 kkg As/hr leaving in blister copper.

z) Based on 0.004 kkg As to stack/hr, fugitive emissions of 0.6 kg As/hr.

aa) Based on 0.006 kkg As sent to dump as slag and acid plant sludge.

bb) Input of 0.01 kkg As/hr.

cc) Based on 0.0004 kkg As/hr leaving in blister copper.

dd) Based on 0.003 kkg As sent to stack/hr, fugitive emissions of 0.8 kg As/hr.

ee) Based on 0.006 kkg As sent to dump as slag and acid plant sludge.

ff) Input of 0.02 kkg As/hr.

gg) Based on 0.0009 kkg As leaving/hr in blister copper

hh) Based on 0.0003 kkg As/hr sent to stack, fugitive emissions of 0.4 kg As/hr.

ii) Based on 0.019 kkg As disposed as slag and acid plant sludge/hr.

jj) Input of 0.068 kkg As/hr.

Table A-3. (Continued)

kk) Based on 0.0039 kkg As/hr leaving in blister copper.

ll) Based on 0.037 kkg As/hr sent to stack, fugitive emission of 2 kg As/hr.

mm) Based on 0.028 kkg As/hr sent to dump as slag and acid plant waste.

nn) Input of 0.004 kkg As/hr.

oo) Based on 0.0001 kkg As/hr leaving in blister copper.

pp) Based on 0.0029 kkg As/hr sent to stack, fugitive emissions of 0.2 kg As/hr

qq) Based on 0.0014 kkg As/hr sent to dump as slag and scrubber waste.

rr) Input of 0.0007 kkg As/hr.

ss) Based on 0.00005 kkg As/hr leaving in blister copper.

tt) BAsed on 0.41 kg As/hr sent to stack, fugitive emissions of 0.05 kg As/hr.

uu) Based on 0.23 kg As/hr sent to dump as slag.

vv) Input of 7.6 kg As/hr.

ww) Based on 0.4 kg As/hr leaving as blister copper.

xx) Based on 6.7 kg As/hr sent to dump as slag and scrubber waste.

yy) Input of 0.045 kg As/hr (0.4 kkg/yr).

zz) Distribution of arsenic not performed.

aaa) Input of 0.84 kkg As/hr.

bbb) Based on 0.2 kkg As/hr leaving in blister copper and stored as dust.

Table A-3. (Concluded)

ccc) Based on 2 kg As/hr sent to stack, fugitive emissions of 0.02 kkg As/hr.

ddd) Based on 0.64 kkg As/hr sent to dump as slag and as sludge to pond.

eee) Based on 3 kkg As/yr/plant discharge, 19 plants and EPA, 1975a control/treatment data. See Appendix A, note 6.

Table A-4. Production, Conversion, and Consumption of Phosphates

	P_2O_5 Quantities, Metric Tons/Year	Arsenic Quantities, Metric Tons/Year
Florida Rock Produced	15,728,300	702
Used as Domestic Fertilizer	8,493,282	382
Used for Animal Feed	552,700	25
Exported	6,569,250	295
Tennessee Rock Produced	617,670	51
For Nonagricultural Uses	617,670	51
Western Rock Produced	1,847,559	425
Used as Domestic Fertilizer	331,610	76
Used for Animal Feed	110,540	25
Exported	110,540	25
For Nonagricultural Uses	1,294,870	298
Total Rock Produced	18,193,530	1,178
Total Used as Domestic Fertilizer	8,824,890	458
Total Used for Animal Feed	663,240	50
Total Exported	6,679,790	320
Total for Nonagricultural Uses	1,912,540	349
Used for Detergents	905,940	160
Used for Food Products	503,300	86
For Miscellaneous Uses	503,300	86

Source: EPA, 1976a, based on 1976 production quantities. Totals do not add due to rounding.

Table A-5. Industrial Wastewaters in which Arsenic has been Detected[a]

Industry	# of Times Detected[b]	# of Samples Taken	% Frequency of Detection
Adhesives/Sealants	2	11	18
Auto/Other Laundries	13	45	29
Coal Mining	31	94	33
Electricial	2	2	100
Foundries	23	54	43
Gum/Wood Products	11	18	61
Inorganic Chemicals	18	107	17
Iron/Steel	145	414	35
Nonferrous Metals	91	146	62
Ore Mining	39	64	61
Organics and Plastics	75	557	13
Paint/Ink	18	149	12
Pesticides	7	104	7
Petroleum Refining	17	346	5
Printing/Publishing	26	66	39
Pulp/Paper	13	44	30
Soap/Detergents	3	20	15
Textile Products	24	121	20
Timber	40	261	15

a) EPA, 1980e. Data for batteries, photographic, phosphates and plastics processing not available.

b) False positives are accepted. Detection limit: 10 µg/l.

Table A-6. Municipal Disposal of Arsenic, 1978 (kkg/yr)

Source	Input	Air	Water	Land
POTW[a]	1,800[b]	neg[c]	1,800[b]	20[d]
Urban runfoff			1,050	
URBAN REFUSE				
Incineration	170[e]	8.7[f]	neg[g]	160[f]
Landfill	3,000[h]			3,000[h]

a) Publicily-owned treatment works. Urban runfoff contribution to POTWs is based on average arsenic concentration of 50 µg/l and a total nationwide flowrate of 2.1×10^{3} l/yr. See Appendix A, Note 21 for derivation of arsenic concentration. EPA 1979j.

b) Assume median effluent 50 µg/l, influent 50 µg/l (see Table A-7). 10^{11} l/day total POTW flow, EPA, 1978c.

c) Atmospheric loss assumed less than 1 kkg.

d) Based on 6×10^{6} kkg dry sludge produced/yr (EPA, 1979i), wet sludge = 95% water (by weight), 170 µg/l as in wet sludge (see Table A-7).

e) Based on 17 g As/kkg in combustible fraction, 10^{7} kkg incinerated yearly, U.S. Dept of Interior, 1978; Gordon, 1978.

f) Based on 0.8 kg As (suspended particulates) emitted/920 kkg solid waste incinerated, 0.8 kg/920 kkg released to land in flyash. The remainder of the land load assumed to come from bottom ash. 10^{7} kkg incinerated yearly. Gordon, 1978, Greenberg et al., 1978; Law and Gordon, 1979.

g) Assumed negligible, by analogy to other heavy metals, Law and Gordon, 1979.

h) 2×10^{8} kkg municipal solid waste, 87% landfilled, 17 ppm As. Geswein, 1980; Alvarez, 1980; U.S. Dept. of Interior, 1978; Gordon, 1978. 17 ppm is concentration in combustible fraction, assumed to represent a minimum for the noncombustible fraction.

Table A-7. Distribution of Arsenic in POTWs and Sludge: Selected Urban Sites[a]

Plant	% Industrial Contribution	Average Flow (10^6 l/day)	Influent Concentration (μg/l)	Influent Loading (kg/day)	Effluent Concentration (μg/l)	Effluent Loading (kg/day)	Combined Sludge Concentration (μg/l)	Combined Sludge Loading (kg/day)
1	30	400	<50	<20	<50	<20	176	1.9
2	<5	30	<50	<1.5	<50	<1.5	149	<0.05
3	10	42	3	0.1	2	0.1	138	<0.05
4	7	320	<50	<16	<50	<16	403	0.3
5	10	83	<50	<4.2	<50	<4.2	78	<0.05
6	38	27	<50	<1.4	<50	<1.4	250	<0.1
7	15	190	<3	<0.6	<3	<0.6	332	0.6
8	10	87	<50	<4.4	<50	<4.4	695	0.5
9	10	200	2	0.4	2	0.4	25	<0.1
10[b]	5	87	ND[c]	ND	ND	ND	27	<0.05
11	18	160	29	4.6	32	5.1	175	0.1
12	50	150	<50	<7.5	<50	<7.5	14	<0.09
13	33	64	<50	<3.2	<50	<3.2	195	2.5
14	25	53	4	0.2	1	0.1	107	<0.05
15	25	27	<50	<1.4	<50	<1.4	1,048	0.05
16	16	550	26	14	<50	14	868	0.6
17	25	57	<50	<2.9	<50	<2.9	282	<0.1
18	50	240	<50	12	<50	12	130	0.3
19	20	260	<50	13	<50	13	167	0.8
20	15-22	450	9	4.1	<50	<23	113	1.1
Mean			31	5.6	37	6.5	269	0.5
Median			50	3.7	50	3.7	171	0.2
Flow Weighted Mean			32		41		302	

a) In calculating mean and median values, those data points designated by < are counted as that upper limit, and considered the "worst case" estimate. Thus, effluent means appear as greater than influent means.

b) It is assumed that the As concentrations in influent and effluent streams were too low to measure, but that the As accumulated to quantifiable levels in sludge.

c) Not detected.

Source: EPA, 1980d.

Table A-8. Industries Contributing to POTWs in Table A-7

Plant	Industry[a]
1	PH, PE, PT, FN, CO, F
2	GS, OFT, MT, MW
3	POP, PL, T
4	BV, PL, PI, CH, F, PA, PP
5	ATO, H, PT, PA, PP
6	PL, FN, BK
7	PT, ATO, FURN
8	ATO
9	FR
10	E, PT, MW, PL
11	E, PT, F
12	PT, F, PH
13	PT, MP, BA
14	PL, PT, PI, PH
15	AP, PL
16	F, MF
17	PI, PT, AR
18	BR, PT, CA, S
19	F, PT, L
20	CA, E, PI, D

Footnotes next page.

Table A-8. (Concluded)

a) Code = the following:

Aircraft Manufacture= AR
Appliance Manufacture = AP
Automobile Manufacture ATO
Baking = BK
Battery Manufacture = BA
Beverages = BV
Breweries = BR
Canneries = Ca
Chemicals = CH
Coking = CO
Detergent Manufacturing = D
Electronics = E
Firearms Manufacture = FR
Foods = F
Foundries = FN
Furniture Manufacture = FURN
Grain Storage = GS
Hospitals = H
Leather Finishing = L
Machine Tools = MT
Meat Packing = MP
Metal Finishing = MF
Metalwork = MW
Oil/Fuel Terminals = OFT
Paint and Ink = PI
Paper = PA
Petrochemicals = PE
Pharmaceuticals = PH
Photo Processing = PP
Plastics = PL
Plating = PT
Poultry processing POP
Slaughterhouse = S
Textiles = T

Table B-1. Arsenical Pesticide Producers and Locations

Pesticide	Use	Producer[d]	Location[d]
MSMA (monosodium methanearsenate)	herbicide[a] citrus fruits cotton crabgrass	Diamond Shamrock Vineland Chem. Co. Inc.	Greens Bayou, TX Vineland, NJ
DSMA (disodium methanearsenate)	herbicide[a] citrus fruits cotton crabgrass	W.A. Cleary Corp. Diamond Shamrock Corp Vineland Chem. Co. In.	Somerset, NJ Greens Bayou, TX Vineland, NJ
Arsenic Acid	Defoliant cotton	Los Angeles, Chem. Co. Osmose Wood Preserving Co. Pennwalt Corp.	South Gate, CA Memphis, TX Bryan, TX
Cacodylic Acid	herbicide[b] lawn control Johnsongrass	Vineland Chem. Co. Inc.	Vineland, NJ
10,10-OBPA (10,10 -oxybisphenoxarsine)	fungicide[c] vinyl plastics	Ventron Corp.	Beverly, MA
Lead Arsenate	insecticide turf ornamentals		
Calcium Arsenat	insecticide cotton vegetables		
Paris Green	insecticide mosquito larva	Los Angeles Chem. Co.	South Gate, CA

a) Directed application, no contact with crop plants (EPA, 1976a).

b) Used only in non crop areas (EPA, 1976a).

c) Also reportedly used as growth regulator on citrus crops, specifically Florida grapefruit (Warshawsky, 1980).

d) L.A. Chem. and W.A. Cleary, according to spokespersons from those companies, no longer produce arsenical pesticides; calcium and lead arsenate are not currently produced in the U.S. (SRI, 1980). Other producer/location data from SRI, 1980.

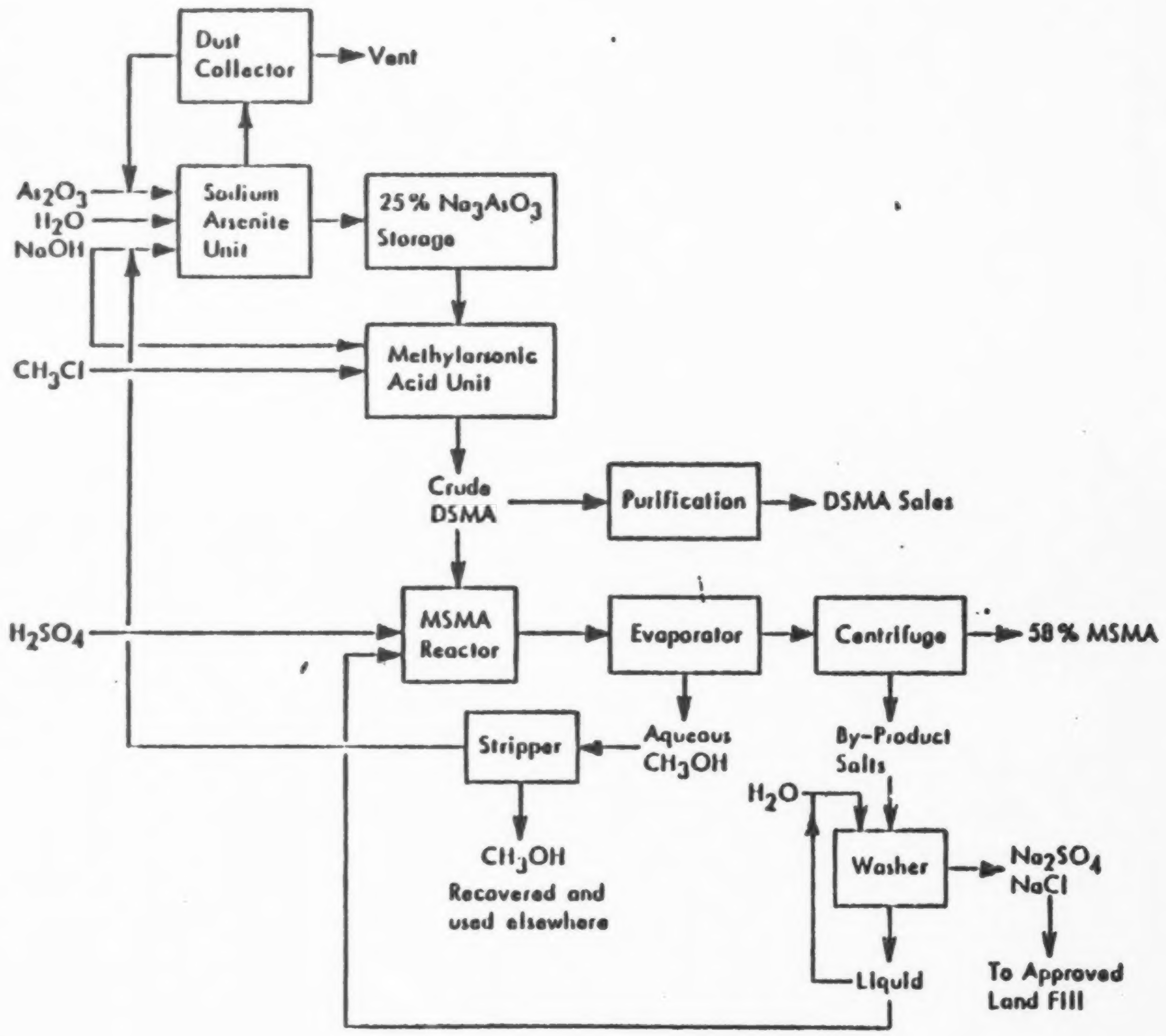
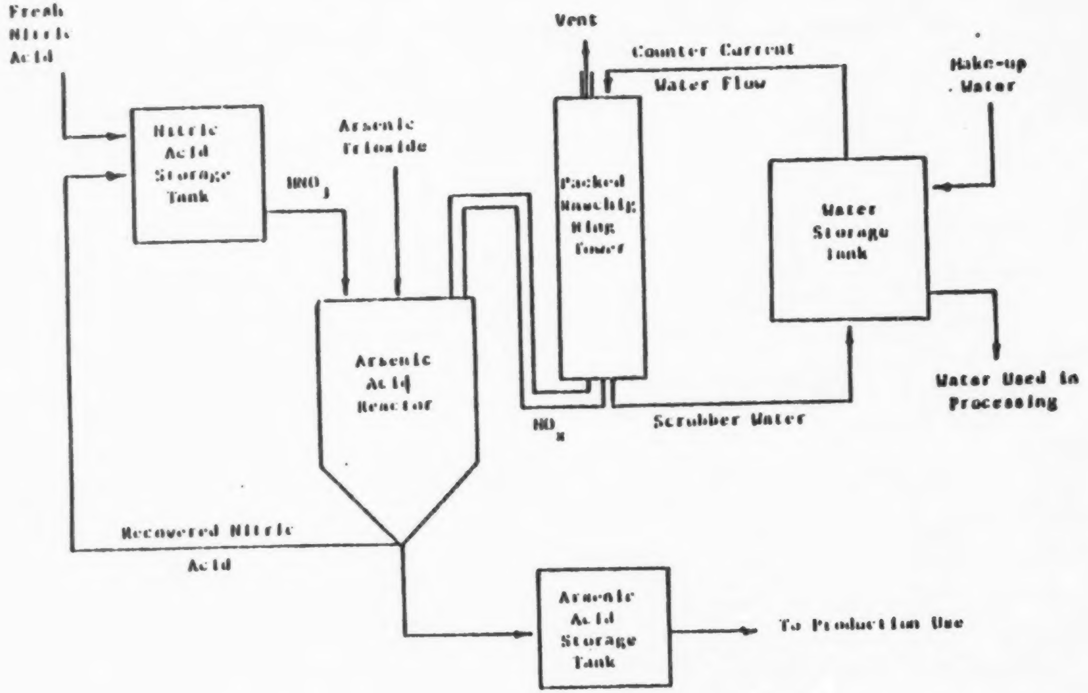
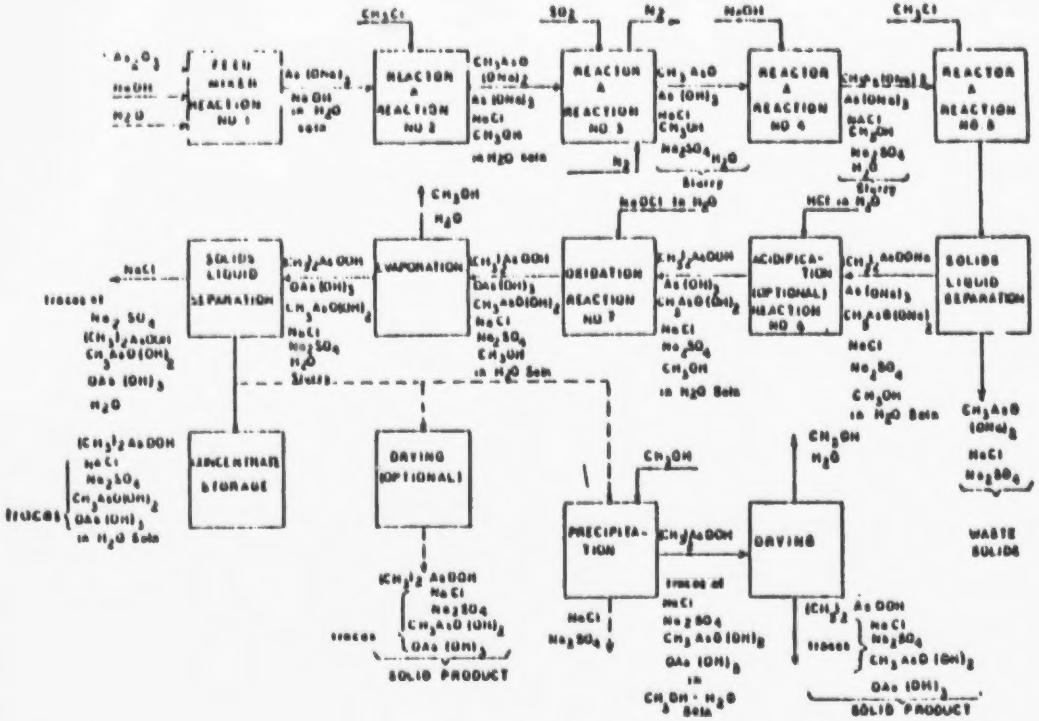

Dust Collector
Vent
As_2O_3
H_2O
NaOH
Sodium Arsenite Unit
25% Na_3AsO_3 Storage
CH_3Cl
Methylarsonic Acid Unit
Crude DSMA
Purification
DSMA Sales
H_2SO_4
MSMA Reactor
Evaporator
Centrifuge
58% MSMA
Stripper
Aqueous CH_3OH
By-Product Salts
H_2O
Washer
Na_2SO_4
NaCl
CH_3OH
Recovered and used elsewhere
Liquid
To Approved Land Fill

Figure B-1. Production Schematic for MSMA and DSMA (EPA, 1974)


Fresh Nitric Acid
Nitric Acid Storage Tank
HNO_3
Arsenic Trioxide
Arsenic Acid Reactor
NO_x
Vent
Packed Raschig Ring Tower
Counter Current Water Flow
Scrubber Water
Water Storage Tank
Make-up Water
Water Used in Processing
Recovered Nitric Acid
Arsenic Acid Storage Tank
To Production Use

Reproduced from best available copy.

Figure B-2. Arsenic Acid Production (Sittig, 1977)



As_2O_3
$NaOH$
H_2O
FEED MIXER REACTION NO 1
$As(ONa)_3$
$NaOH$ in H_2O soln
CH_3Cl
REACTION A REACTION NO 2
$CH_3AsO(ONa)_2$
$As(ONa)_3$
$NaCl$
CH_3OH
in H_2O soln
SO_2
N_2
REACTOR A REACTION NO 3
$CH_3AsO(OH)_2$
$As(OH)_3$
$NaCl$
CH_3OH
Na_2SO_4, H_2O Slurry
$NaOH$
REACTOR A REACTION NO 4
$(CH_3)_2AsO(ONa)$
$As(ONa)_3$
$NaCl$
CH_3OH
Na_2SO_4
H_2O
Slurry
CH_3Cl
REACTOR A REACTION NO 5
$NaOCl$ in H_2O
HCl in H_2O
SOLIDS LIQUID SEPARATION
$(CH_3)_2AsOONa$
$As(ONa)_3$
$CH_3AsO(ONa)_2$
$NaCl$
Na_2SO_4
CH_3OH
in H_2O Soln
ACIDIFICATION (OPTIONAL) REACTION NO 6
$(CH_3)_2AsOOH$
$As(OH)_3$
$CH_3AsO(OH)_2$
$NaCl$
Na_2SO_4
CH_3OH
in H_2O Soln
OXIDATION REACTION NO 7
$(CH_3)_2AsOOH$
$OAs(OH)_3$
$CH_3AsO(OH)_2$
$NaCl$
Na_2SO_4
CH_3OH
in H_2O Soln
CH_3OH
H_2O
EVAPORATION
$(CH_3)_2AsOOH$
$OAs(OH)_3$
$CH_3AsO(OH)_2$
$NaCl$
Na_2SO_4
H_2O
Slurry
SOLIDS LIQUID SEPARATION
$NaCl$
traces of
Na_2SO_4
$(CH_3)_2AsOOH$
$CH_3AsO(OH)_2$
$OAs(OH)_3$
H_2O
CONCENTRATE STORAGE
$(CH_3)_2AsOOH$
$NaCl$
Na_2SO_4
$CH_3AsO(OH)_2$
$OAs(OH)_3$
in H_2O Soln
traces
DRYING (OPTIONAL)
$(CH_3)_2AsOOH$
$NaCl$
Na_2SO_4
$CH_3AsO(OH)_2$
$OAs(OH)_3$
traces
SOLID PRODUCT
CH_3OH
PRECIPITATION
$NaCl$
Na_2SO_4
$(CH_3)_2AsOOH$
traces of
$NaCl$
Na_2SO_4
$CH_3AsO(OH)_2$
$OAs(OH)_3$
in
$CH_3OH \cdot H_2O$ Soln
DRYING
CH_3OH
H_2O
$(CH_3)_2AsOOH$
traces
$NaCl$
Na_2SO_4
$CH_3AsO(OH)_2$
$OAs(OH)_3$
SOLID PRODUCT
$CH_3AsO(ONa)_2$
$NaCl$
Na_2SO_4
WASTE SOLIDS

Figure B-3. Block Flow Diagram of Process for Cacodylic Acid Manufacture (Sittig, 1977)

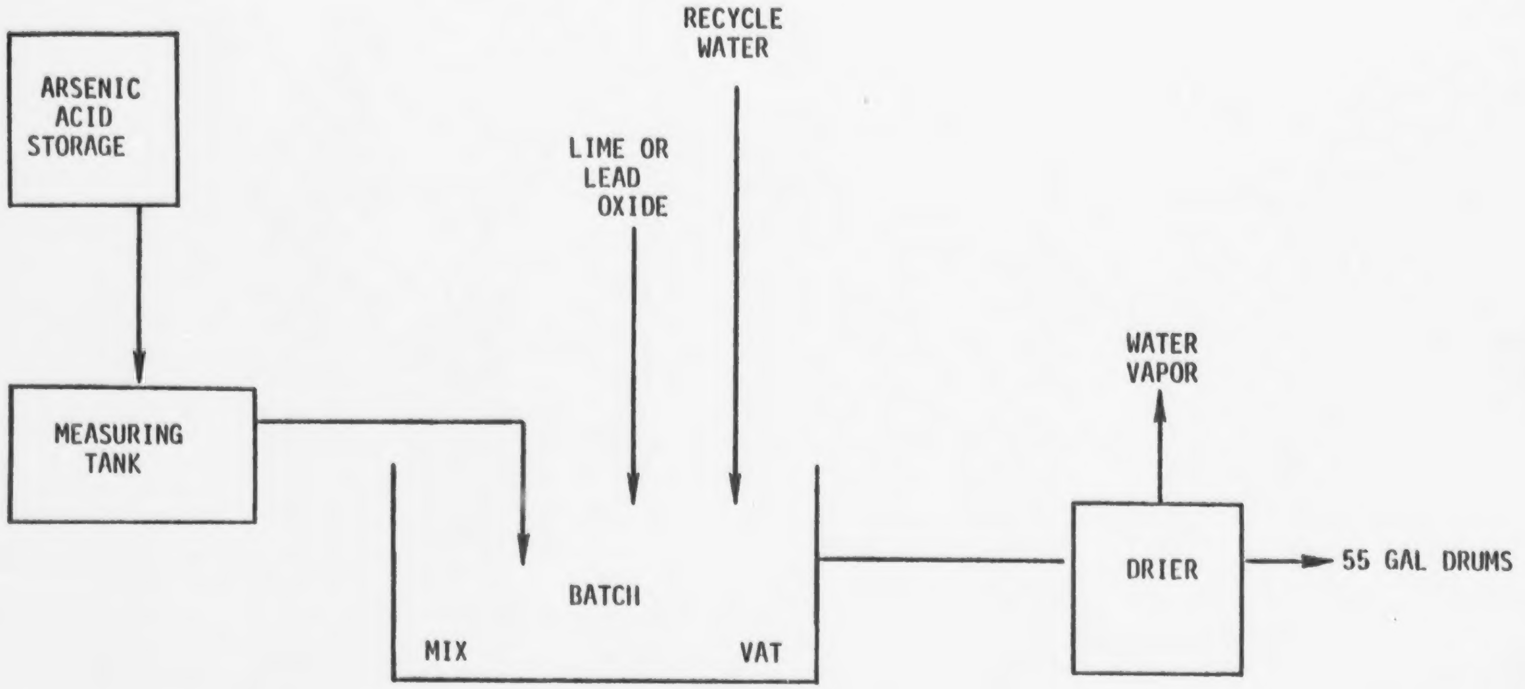

ARSENIC ACID STORAGE
MEASURING TANK
LIME OR LEAD OXIDE
RECYCLE WATER
BATCH
MIX
VAT
WATER VAPOR
DRIER
55 GAL DRUMS

Figure B-4. Production of Lead and Calcium Arsenate (Sittig, 1977)

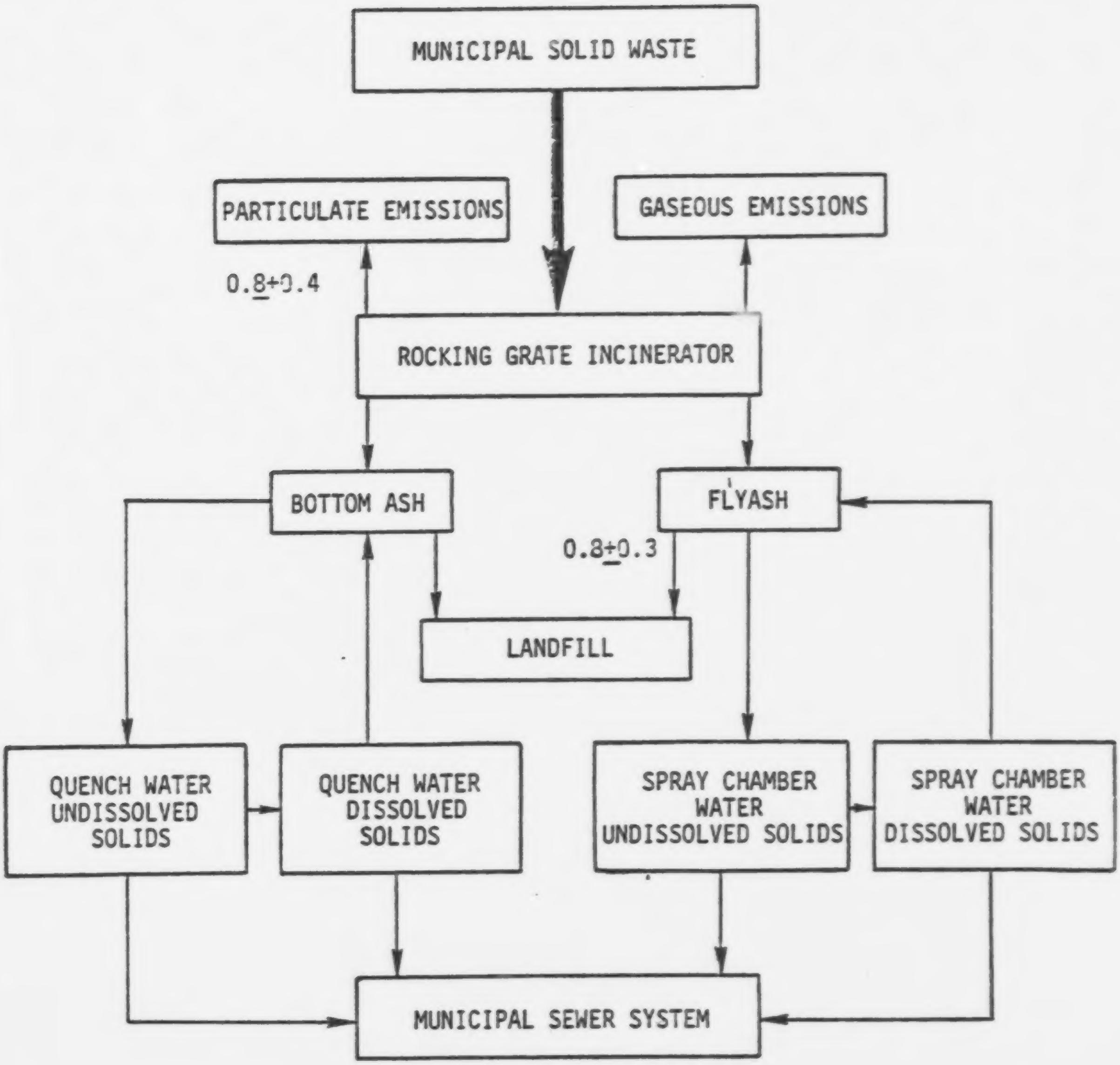

MUNICIPAL SOLID WASTE
PARTICULATE EMISSIONS
GASEOUS EMISSIONS
0.8±0.4
ROCKING GRATE INCINERATOR
BOTTOM ASH
FLYASH
0.8±0.3
LANDFILL
QUENCH WATER UNDISSOLVED SOLIDS
QUENCH WATER DISSOLVED SOLIDS
SPRAY CHAMBER WATER UNDISSOLVED SOLIDS
SPRAY CHAMBER WATER DISSOLVED SOLIDS
MUNICIPAL SEWER SYSTEM

Figure B-5. Flow Diagram of a Municipal Incinerator in Kilograms Arsenic (Law and Gordon, 1979)

---

a) kg arsenic per 920 kkg refuse.

APPENDIX C

This distribution of arsenic emissions and discharges from combustion of coal and oil* for energy production is derived from the following basic assumptions.:

(1) The ash present in coal is distributed between bottom ash, flyash collected, and particulate stack emissions (i.e., ash loss via slagging is negligible); and

(2) the distribution of the arsenic originally present in the feed material is dependent only upon particle size.

Thus: Ash in (A_{in}) = Ash out (A_{out}) = $A_b + A_c + A_e$, or

$$1 = A_{in} = A_b + A_c + A_e$$

where A_b = Fraction Bottom ash

A_c = Fraction Flyash collected

A_e = Fraction Flyash emitted, also,

$A_c + A_e = A_f$ where A_f = Fraction of ash that is total flyash.

The amount of ash which appears as bottom ash, is dependent upon fuel and boiler type. For boilers which fire pulverized coal (<1 cm) eighty percent of the ash originally present in coal is estimated to appear as flyash; ash produced in cyclone boilers, which burn a somewhat larger size of coal than pulverized coal fired units, is distributed about equally between bottom ash and flyash; of the ash produced in stoker fired boilers, which burn relatively large sizes of coal (>10 cm), approximately twenty-five percent appears as flyash.

---

*Though the derivation is applicable to gas fired boilers as well, natural gas contains negligible amounts of arsenic and thus is an insignificant source of arsenic emissions to the environment (Slater and Hall, 1977).

Essentially all of the ash present in residual oil appears as flyash. The amount of flyash collected in turn depends upon the particulate control device(s) used, which generally are cyclonic devices, electrostatic precipitators, or baghouses. The fraction of ash emitted as flyash, control device efficiency, application of control, effective efficiency, fine particulate fraction, atmospheric emission fraction, and land dispersion fraction are shown in Table C1. For pulverized coal fired boilers, ash emission fractions are calculated as follows:

$$(1-A_f) = A_b = 0.20$$

$$A_c = (A_f)E = 0.71$$

$$A_e = (A_f)(1-E) = 0.088$$

The fraction of the ash emitted to the atmosphere (A_e) is further subdivided into particulate which remains suspended (A_e <3 μm) and that which eventually settles to land (A_e >3 μm) in the following way:

$$A_e > 3\mu = A_e P_f = 0.057$$

$$A_e < 3\mu = A_e(1-P_f) = 0.031$$

Arsenic emissions and discharges within a boiler are a function of particle size, arsenic concentration increasing with decreasing particle size. Arsenic concentration of flyash by particle size ranges is shown in Table C2. Using these data (and assuming the arsenic concentration of bottom ash and collected flyash to be equal) relative arsenic concentrations of bottom ash (C_b), collected flyash (C_c), flyash emitted >3μ (C_e <3μ), and flyash emitted

$<3\mu$ $(C_e>3\mu)$ are calculated to be 1.0, 1.0, 3.3, 5.7, respectively.

Arsenic emission factors are calculated in the following way.

$$1 = f_b + f_c + f_e <3\ \mu m + f_e >3\ \mu m$$

where:

f_b = Fraction of arsenic contained in bottom ash

f_c = Fraction of arsenic contained in collected flyash

$f_e<3\mu$ = Fraction of arsenic emitted as particulate $<3\mu$

$f_e>3\mu$ = Fraction of arsenic emitted as particulate matter $>3\mu$.

These fractions are calculated using ash emission fractions and relative arsenic concentrations:

$$As_{total} = A_b \bullet C_b + A_c \bullet C_c + (A_{e<3\mu} \bullet C_{e<3\mu}) + (A_{e>3\mu} \bullet C_{e>3\mu})$$

$$f_b = \frac{A_b \bullet C_b}{As_{total}}$$

$$f_c = \frac{A_c \bullet C_c}{As_{total}}$$

$$f_e<3\mu m = \frac{A_{e<3\mu m}\ (C_{e<3\mu m})}{As_{total}}$$

$$f_e>3\mu m = \frac{A_{e>3\mu m}\ (C_{e>3\mu m})}{As_{total}}$$

Consequently, arsenic emission factors by boiler type and media are shown in Table C3.

Table C-1. Arsanic Wastes: Energy Production (kkg/yr)

Source	Fuel Consumption[a]	Total Arsenic[b]	Dispersion[c] Air Total	 <3 µm	 >3 µm	Land[d]	Water
PRODUCTION						----	
Coal	617,600,000	16,450					95[f]
Bituminous	580,733,000	16,260				neg[e]	
Anthracite	949,000	10					
Lignite	34,354,000	175					
Petroleum	909,300,000	76					
Residual	154,600,000	46					
Distillate	754,082,000	30					
ELECTRICITY GENERATION							
External Combustion							
Coal (total)	479,600,000	12,590	3,530	1,750	1,780	10,790	52[g]
Bituminous (total)	442,733,000	12,400	3,490	1,725	1,760	10,640	
Pulverized dry	336,477,000	9,420	2,730	1,320	1,410	8,100	
Pulverized wet	42,733,000	1,200	350	170	180	1,030	
Cyclone	53,128,000	1,490	340	220	120	1,270	
All stokers	8,855,000	250	65	15	50	240	
Anthracite (total)	949,000	10	2	<1	2	10	
Pulverized dry	----						
Pulverized wet	----						
Cyclone	----						
All stokers	949,000	10	2	<1	2	10	
Lignite (total)	34,354,000	170	45	25	20	150	
Pulverized dry	12,367,000	63	19	9	10	54	
Pulverized wet	2,405,000	12	4	2	2	10	
Cyclone	18,210,000	93	21	14	7	79	
All stokers	1,031,000	5	1	<1	1	5	
Petroleum	77,850,000	24	24	23	1	1	neg[h]
Residual oil	72,230,000	22	22	21	1	1	
Combustion turbine	83,600	neg	neg			neg[h]	
Steam generation	71,910,000	22	22	21	1	1	
Combined cycle	328,600	neg	neg			neg[h]	
Distillate oil	5,582,000	2	2	2	neg	neg[h]	
Combustion turbine	3,430,000	2	2	2	neg	neg[h]	
Stream generation	1,484,000						
Combined cycle	667,500						
SPACE HEATING AND OTHER							
Coal[f]		3,860	1,000	230	770	3,630	neg
Industry	60,000,000	1,580	440	100	340	1,580	
Residential/Commercial	8,000,000	220	54	10	44	210	
Coke plants	70,000,000	1,960	510	120	390	1,840	
Petroleum	240,000,000	54	54	52	2	2	neg
Residual oil	82,400,000	24	24	23	1	1	
Industry	61,100,000	18	18	17	1	1	
Residential/Commercial	neg	neg	neg			neg	
Transportation	21,300,000	6	6	6	neg	neg	
Distillate oil	748,500,000	30[h]	30[h]	29	1	1[h]	
Industry	80,000,000	3	3	3	neg	neg	
Residential/Commercial	189,000,000	8[h]	8[h]	8	neg	neg[h]	
Transportation	479,500,000	19	13	13	1	1	

Table C-1. (Concluded)

a) Production and consumption data are from Monthly Energy Review published by National Reliability Council, Princeton, N.J. and are rounded to nearest thousand metric ton. Data may not add due to rounding.

b) Arsenic concentration by coal type is assumed to be: Bituminous coal - 28 mg/kg; Anthracite - 10 mg/kg; Lignite - 5.1 mg/kg. By oil type: Residual - 0.3 mg/kg; Distillate - 0.04 mg/kg (Slater and Hall, 1977).

c) The amount of arsenic emitted to the atmosphere was calcualted as follows:

Q=(C)x(F)
Where Q=quantity of arsenic in feed material
C=concentration of arsenic in fuel, ppm
F=yearly consumption of fuel, metric tons per year

The amount emitted to the atmosphere, E, was
E=(Q)x (fe <3 μm)

Where fe <3 μm is calculated in Appendix C; arsenic associated with particles that are emitted to the atmosphere and are greater than 3 μm in diameter settle out quickly and are thus included with land waste

d) The amount of arsenic discharged to land was calculated as follows:

L=(Q-E)+(E >3 μm)
Where L=amount of arsenic discharged to land:
Q=amount of arsenic in fuel;
E=amount of arsenic emitted to the atmosphere
E > 3μm=amount of arsenic associated with particles greater than 3 μm in diameter and that were initially emitted to the atmosphere, but eventually settled to land

e) National particulate emission burden from coal storage piles is estimated to be 630 kkg/yr; Blackwood and Wacher (1978). Arsenic fugitive emissions are calculated using the following arsenic concentrations: Bituminous coal - 28 mg/kg; Anthracite - 10 mg/kg; Lignite - 5.1 mg/kg. Neg = <1 kkg.

f) Acid mine drainage is reported to average 12 mg/kg (28) at an average flow of 3.8 x 10^3 liters per mine-day. Drainage from 5673 coal mines is considered in this calculation.

g) Screening sampling data for the steam electric power point source category: average flow, wet fly ash - 9.9 x 10^6 1/day/plant, wet bottom ash - 9.8 x 10^6 1/day/plant; 183 plants (52% of all coal plants) use wet fly ash transport and 219 plants use wet bottom ash transport; average concentrations: bottom pond effluent, 0.016 mg/1, fly ash pond effluent, 0.060 mg/1. EPA, 1980; FR 1980.

h) Neg = <1 kkg.

i) Coal combustion exclusive of utilities is assumed to be in stoker fired boilers.

Table C-2. Mass Efficiency of Particulate Collection on Utility Boilers[a]

Fuel	Boiler Type	Fraction of[b] Ash Emitted As Flyash (A_F)	Control Device Efficiency[c]	Application of Control[d]	Effective Efficiency		Fine Particulate Fraction (<3 μm) (P_f)	Atmospheric Emission[h]		Land Dispersion[h]	
					Calculated	Reported[f] (E)		(<3 μm) Ae <3 μm	(>3 μm) Ae >3 μm	Flyash	Bottom Ash
Coal	Pulverized	0.8	0.92	0.97	0.89	0.89	0.35	0.031	0.057	0.71	0.20
	Cyclone	0.50	0.91	0.71	0.65	0.88	0.52	0.031	0.029	0.44	0.50
	Stoker	0.25	0.80	0.87	0.70	0.65	0.14	0.012	0.075	0.16	0.75
Oil	All	1.0	0.50	0.20	0.10	0.10	0.90	0.81	0.19	-----	-----
Gas	All	1.0	0	0	0		0.90	0.90	0.10	-----	-----

a) Data were obtained from utilities accounting for one-half of the total U.S. utility consumption of coal in 1974.

b) Engineering estimates based upon published data.

c) Fraction collected of the total particulate mass entering control devices. Data includes both test results and reported design efficiencies.

d) Fraction of utility boiler equipped with particulate control devices.

e) Effective efficiency equals control device efficiency multiplied by application of control.

f) Slater and Hall, 1977.

g) Fine particulate fraction is the fraction of total particulate mass emission <3 μm, derived from particulate size distribution entering control device; the effective mass efficiency; and the effective fine particulate efficiency.

h) Fraction emitted based upon reported effective efficiency, fine particulate fraction, and distribution of coal ash between flyash and bottom ash. For pulverized and cyclone boilers, the distribution fraction between flyash and bottom ash (engineering estimates based upon unpublished data) is estimated to be 0.8 and 0.2 respectively; for stoker fired boilers, the distribution fraction between flyash and bottom ash is estimated to be 0.2 and 0.8 respectively.

Source: Slater and Hall, 1977.

Table C-3. Arsenic Concentration in Coal Flyash as a Function of Particle Size

Particle Diameter (µm)	Arsenic Concentration µg/g	Mass Fraction (%)
Flyash retained in the plant Sieved fraction:		
>74	180	66.30
44-74	500	22.89
Aerodynamically sized fractions		
>40	120	2.50
30.40	160	3.54
20-30	200	3.25
15-20	300	0.80
10-15	400	0.31
5-10	800	0.33
<5	370	0.08
Airborne flyash[a]		
>11.3	680	
7.3-11.3	800	
4.7-7.3	1,000	
3.3-4.7	900	
2.1-3.3	1,200	
1.1-2.1	1,700	

a) An equal mass distribution among particulate size fractions is assumed.

Source: EPA, 1980c.

Table C-4. . Arsenic Emission Factors by Boiler Type and Media

Fuel	Boiler Type	Arsenic Distribution[a]			
		Bottom Ash (f_b)	Flyash Captured (f_c)	Flyash Emitted (<3 μm) ($f_{e <3\ \mu m}$)	Flyash Emitted (>3 μm) ($f_{e >3\ \mu m}$)
Coal	Pulverized	0.16	0.56	0.14	0.15
	Cyclone	0.41	0.36	0.15	0.08
	Stoker	0.61	0.13	0.06	0.20
Oil	All	----	----	0.96	0.04

a) Fraction emitted based upon ash distribution and relative arsenic concentration of flyash <3μ, flyash >3μ, flyash collected, and bottom ash. The concentration ratios are (see Table C2 also):

flyash emitted (<3 μ) = 5.7

flyash emitted (>3 μ) = 3.3

flyash collected = 1.0

bottom ash = 1.0

APPENDIX D

GEOGRAPHIC DISTRIBUTION OF INDUSTRIAL SOURCES OF ARSENIC RELEASES

This appendix consists of maps and tables describing the national distribution of the most significant industrial sources of arsenic releases to the natural environment. Included are the locations of inadvertent sources such as mining and smelting operations and the regional characteristics of arsenic pesticide use. Also included are the national distribution of selected climatological or environmental properties having a potential influence on the potential for arsenic mobility in soil. Most of the information is from U.S. EPA (1980c) unless otherwise noted.

The following maps and tables are included within this appendix:

Figure D-1 Location of Producers of Sodium and Its Derivatives DSMA, MSMA and Cacodylic Acid

Figure D-2 Location of Producers of Arsenic Acid and Its Derivatives

Figure D-3 Geographical Distribution of Wood Preserving Plants in the United States

Figure D-4 Location of Arsenic Acid Use in Texas Cotton Areas

Figure D-5 Location of the Major Coal Burning Power Plants in the United States

Figure D-6 Locations of Primary Copper, Lead, and Zinc Smelters

Figure D-7 Lead Mines and Smelters in the United States

Figure D-8 Geographical Distribution of the Iron and Steel Industry

Figure D-9 Location Map of the Primary Aluminum Industry in the United States

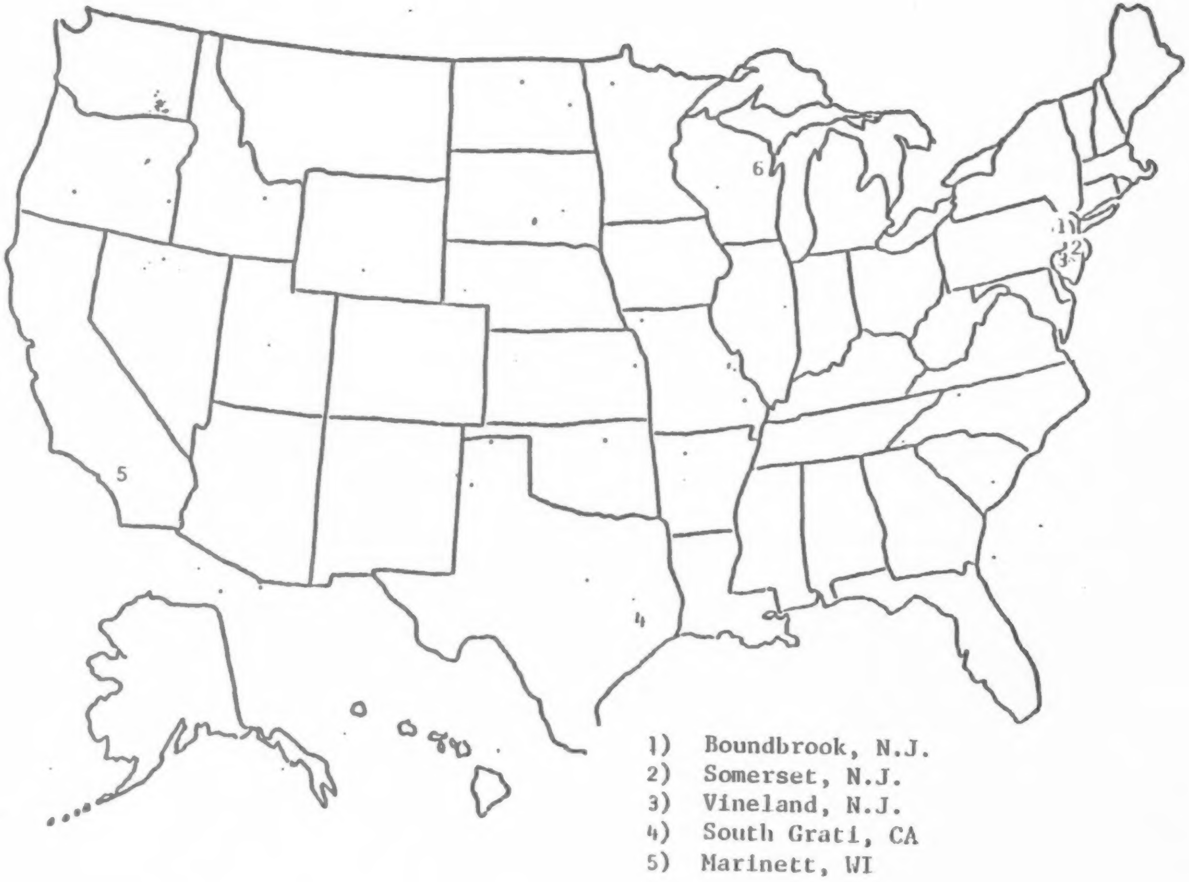
Figure D-10 Erosion Map of the United States

Figure D-11 Soil Scenario Regions

Figure D-12 Distribution of Precipitation

Table D-1 Estimated Uses of MSMA in the United States by Regions and Categories, 1979

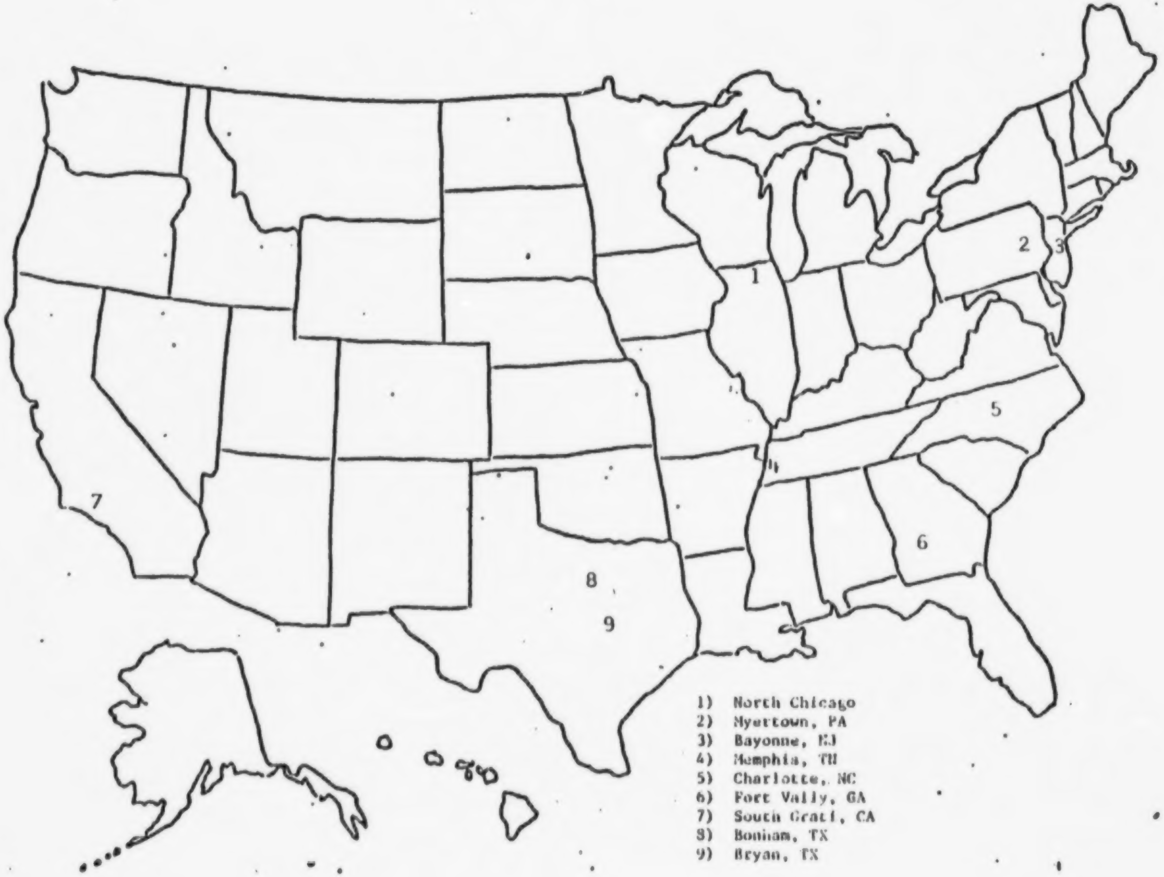
Table D-2 Companies Engaged in Boron Production



1) Boundbrook, N.J.
2) Somerset, N.J.
3) Vineland, N.J.
4) South Grati, CA
5) Marinett, WI

FIGURE D-1 LOCATION OF PRODUCERS OF SODIUM ARSENITE AND ITS DERIVATIVES DSMA, MSMA AND CACODYLIC ACID

Source; U. S. EPA 1980c



1
2
3
4
5
6
7
8
9
1) North Chicago
2) Myertown, PA
3) Bayonne, NJ
4) Memphis, TN
5) Charlotte, NC
6) Fort Vally, GA
7) South Grati, CA
8) Bonham, TX
9) Bryan, TX

FIGURE D-2 LOCATION OF PRODUCERS OF ARSENIC ACID AND ITS DERIVATIVES

Source: U. S. EPA 1980c

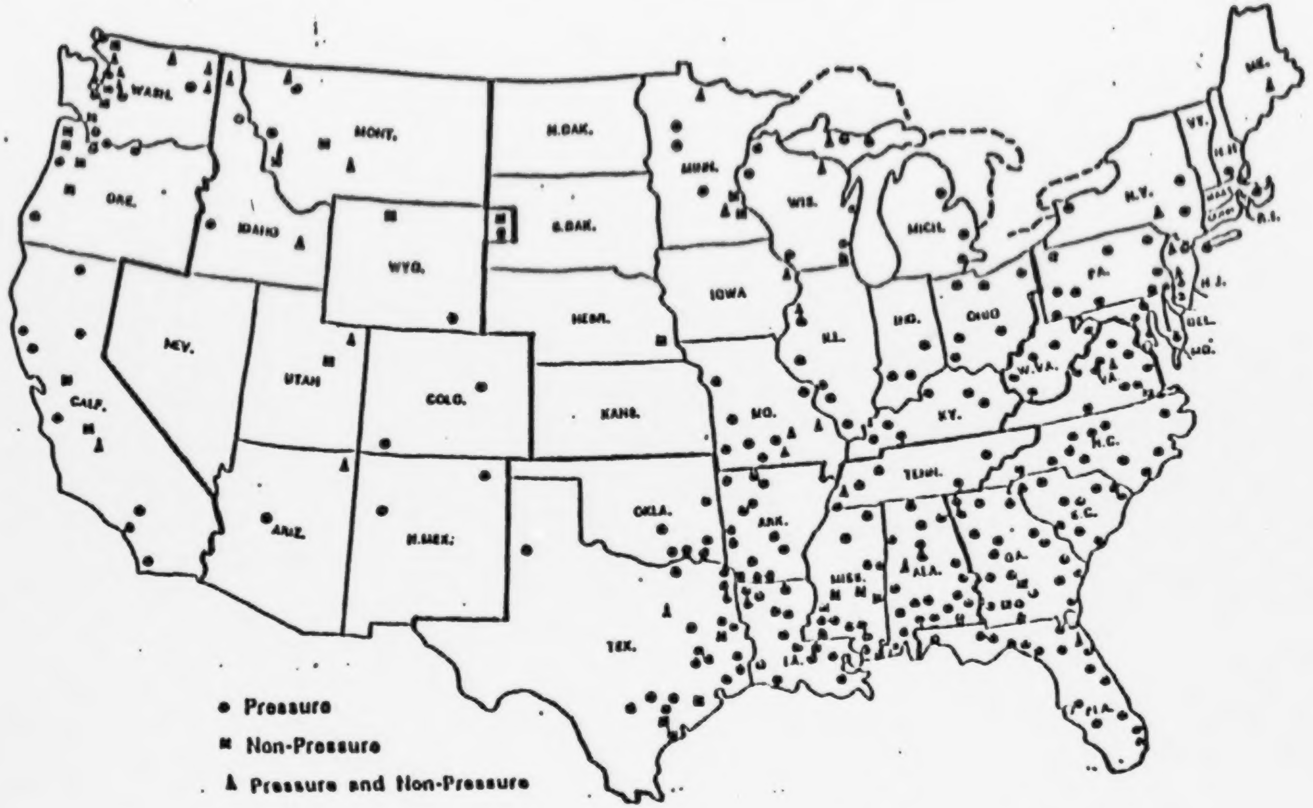

WASH.
ORE.
CALF.
IDAHO
MONT.
WYO.
NEV.
UTAH
COLO.
ARIZ.
N.MEX.
N.DAK.
S.DAK.
NEBR.
KANS.
OKLA.
TEX.
MINN.
IOWA
MO.
ARK.
LA.
WIS.
ILL.
MICH.
IND.
OHIO
KY.
TENN.
MISS.
ALA.
GA.
S.C.
N.C.
VA.
W.VA.
PA.
N.Y.
VT.
N.H.
ME.
MASS.
CONN.
R.I.
N.J.
DEL.
MD.
FLA.
● Pressure
■ Non-Pressure
▲ Pressure and Non-Pressure

FIGURE D-3 GEOGRAPHICAL DISTRIBUTION OF WOOD PRESERVING PLANTS IN THE UNITED STATES

Source: U.S. EPA 1980c

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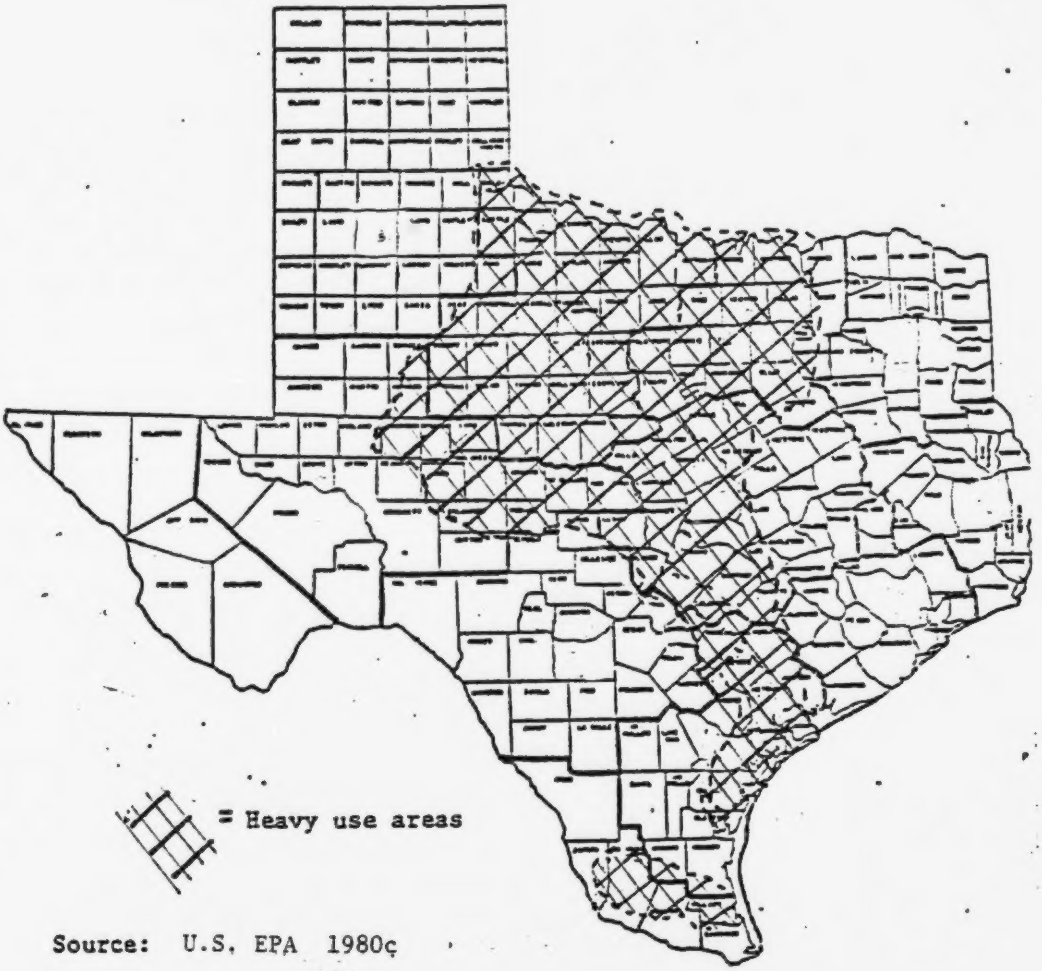
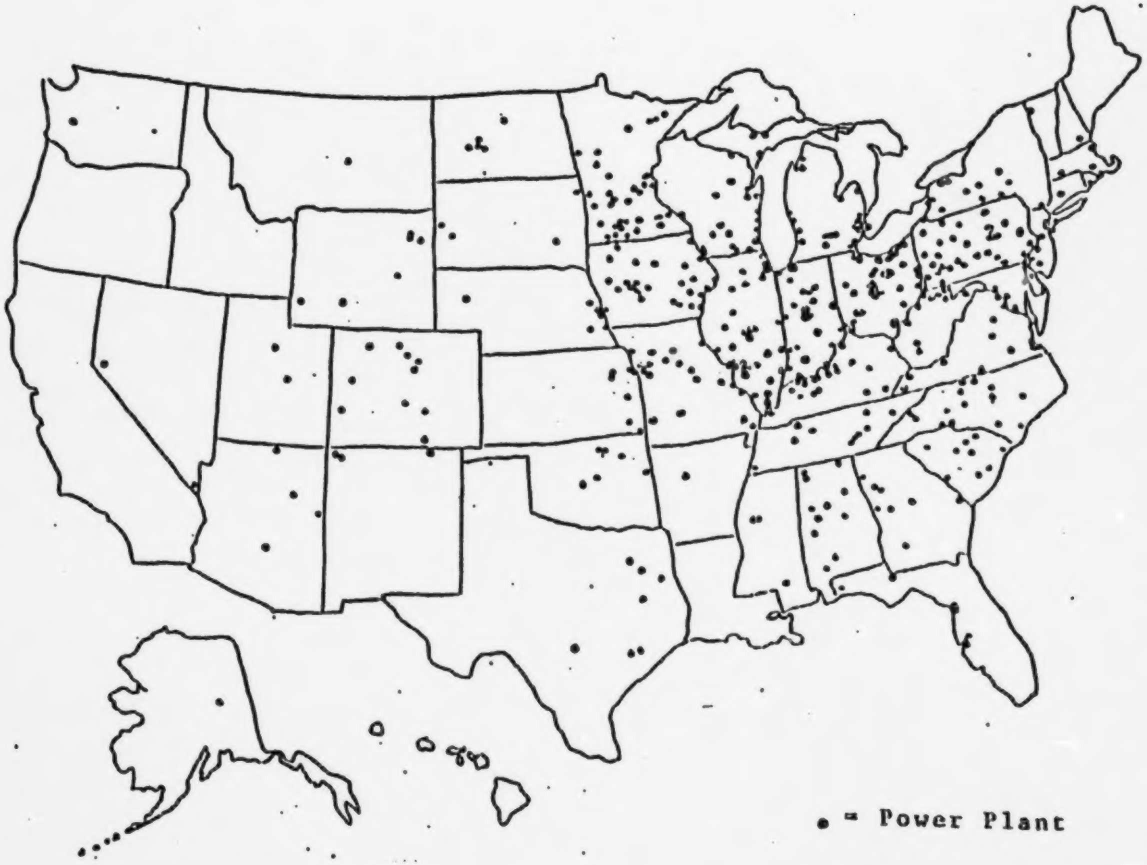
best available copy.


= Heavy use areas

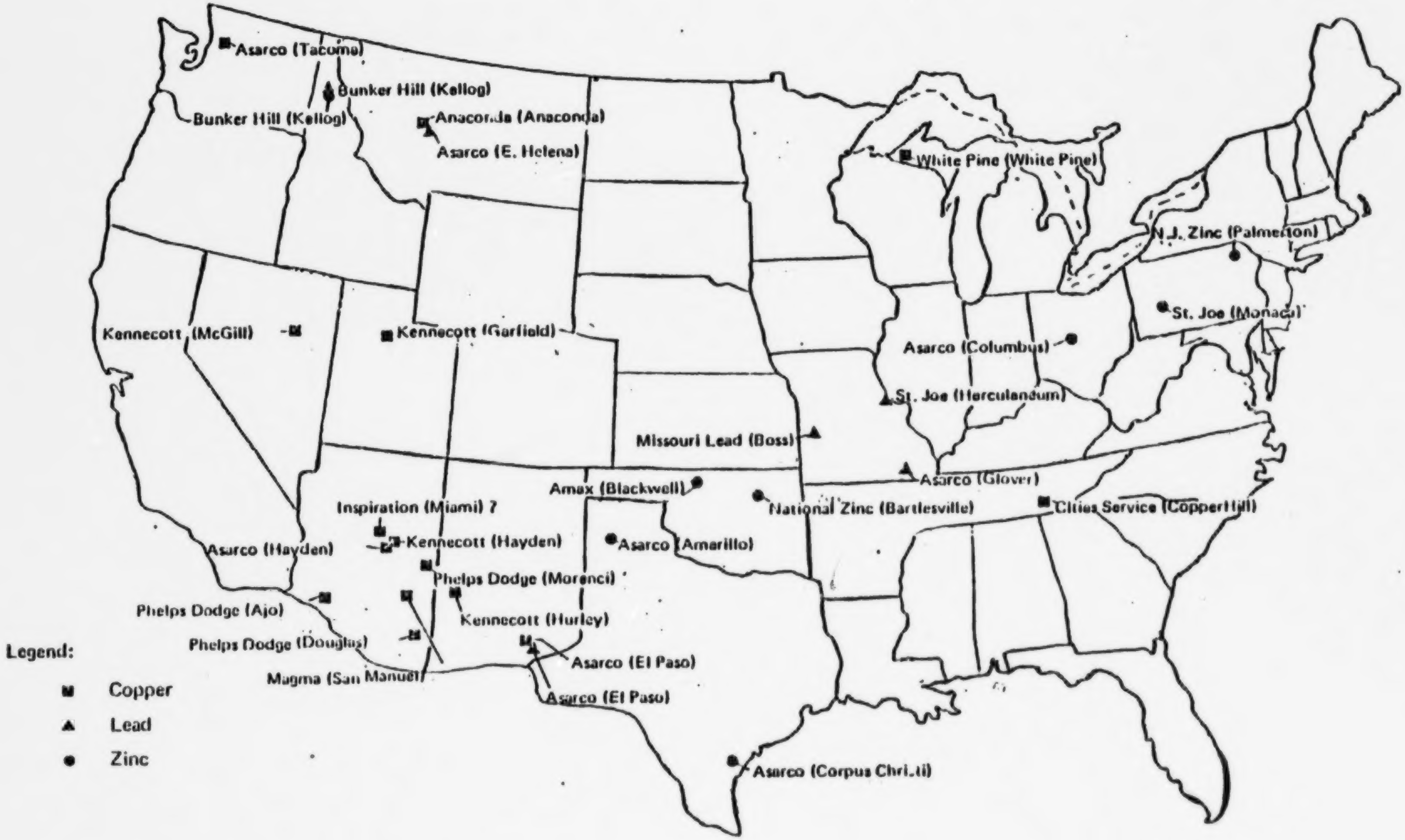
Source: U.S. EPA 1980c

FIGURE D-4 LOCATION OF ARSENIC ACID USE
IN TEXAS COTTON AREAS


• = Power Plant

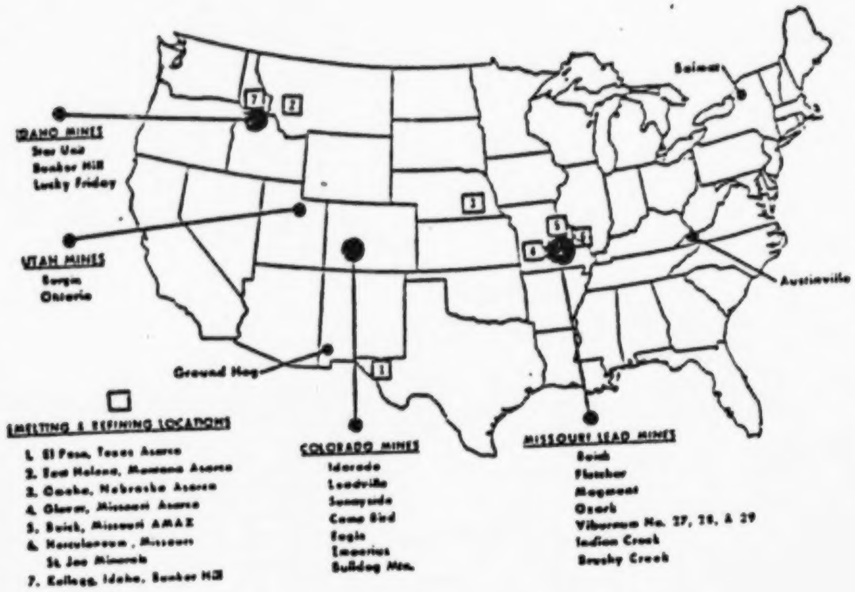
Source: U.S. EPA 1980c

FIGURE D-5 LOCATION MAP OF THE MAJOR COAL BURNING POWER PLANTS IN THE UNITED STATES


Asarco (Tacoma)
Bunker Hill (Kellog)
Bunker Hill (Kellog)
Anaconda (Anaconda)
Asarco (E. Helena)
White Pine (White Pine)
N.J. Zinc (Palmerton)
St. Joe (Monaca)
Kennecott (McGill)
Kennecott (Garfield)
Asarco (Columbus)
St. Joe (Herculandum)
Missouri Lead (Boss)
Asarco (Glover)
Amax (Blackwell)
National Zinc (Bartlesville)
Cities Service (Copperhill)
Inspiration (Miami) 7
Kennecott (Hayden)
Asarco (Hayden)
Asarco (Amarillo)
Phelps Dodge (Morenci)
Phelps Dodge (Ajo)
Kennecott (Hurley)
Phelps Dodge (Douglas)
Asarco (El Paso)
Magma (San Manuel)
Asarco (El Paso)
Asarco (Corpus Christi)
Legend:
■ Copper
▲ Lead
● Zinc

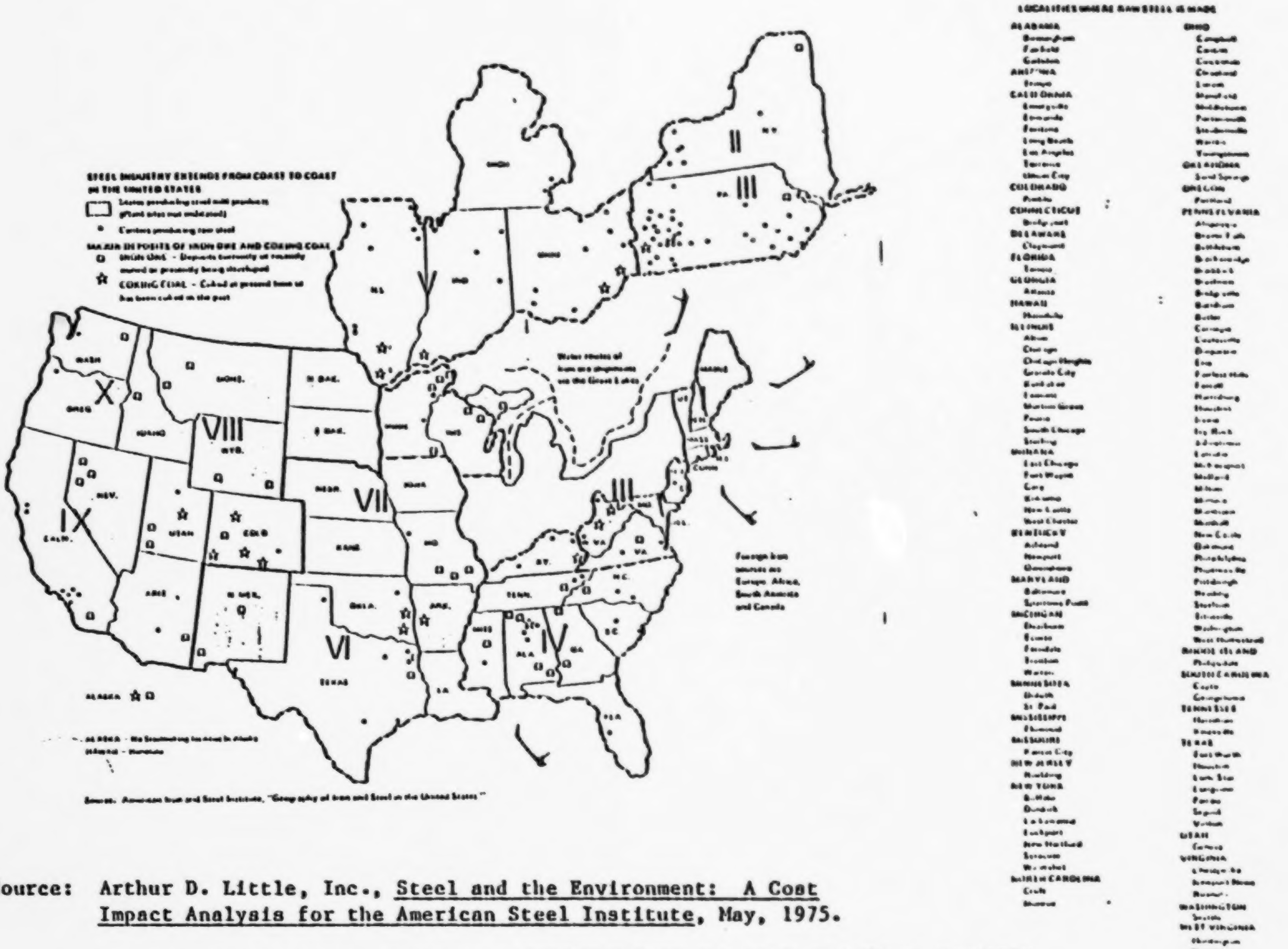
Source: U.S. EPA 1980c

FIGURE 6-D LOCATIONS OF PRIMARY COPPER, LEAD, AND ZINC SMELTERS


IDAHO MINES
Star Unit
Bunker Hill
Lucky Friday
UTAH MINES
Burgin
Ontario
Ground Hog
Balmat
Austinville
SMELTING & REFINING LOCATIONS
1. El Paso, Texas Asarco
2. East Helena, Montana Asarco
3. Omaha, Nebraska Asarco
4. Glover, Missouri Asarco
5. Buick, Missouri AMAX
6. Herculaneum, Missouri St. Joe Minerals
7. Kellogg, Idaho, Bunker Hill
COLORADO MINES
Idarado
Leadville
Sunnyside
Camp Bird
Eagle
Emperius
Bulldog Mtn.
MISSOURI LEAD MINES
Buick
Fletcher
Magmont
Ozark
Viburnum No. 27, 28, & 29
Indian Creek
Brushy Creek
1
2
3
4
5
6
7

Source: U. S. EPA 1980c

FIGURE D-7 LEAD MINES AND SMELTERS IN THE UNITED STATES


STEEL INDUSTRY EXTENDS FROM COAST TO COAST IN THE UNITED STATES
COKING COAL
LOCALITIES WHERE RAW STEEL IS MADE
I
II
III
IV
V
VI
VII
VIII
IX
X

Source: Arthur D. Little, Inc., Steel and the Environment: A Cost Impact Analysis for the American Steel Institute, May, 1975.

FIGURE D-8 GEOGRAPHICAL DISTRIBUTION OF THE IRON AND STEEL INDUSTRY

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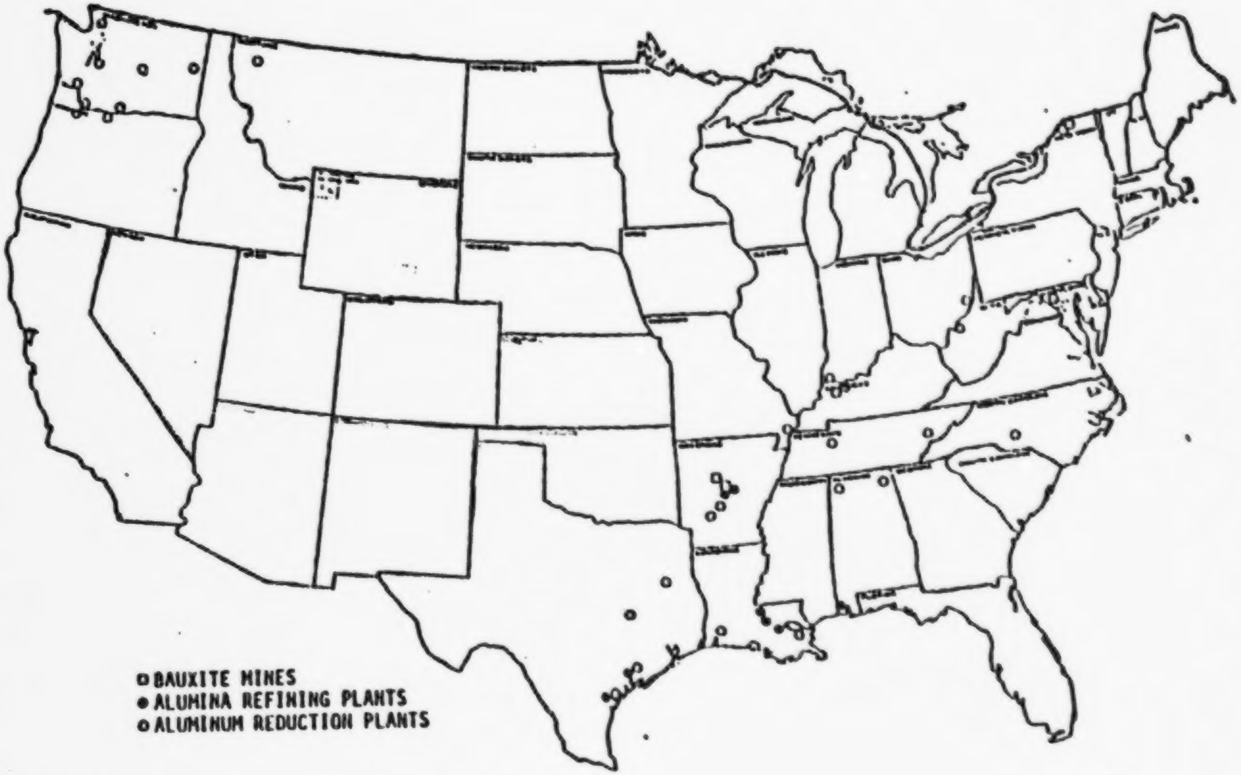
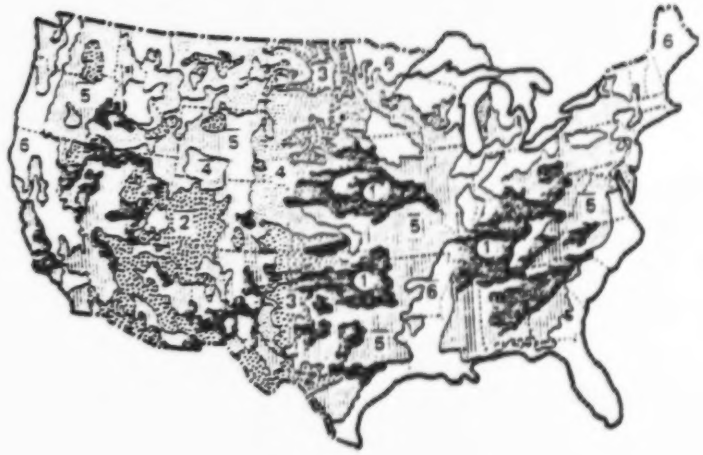

□ BAUXITE MINES
● ALUMINA REFINING PLANTS
○ ALUMINUM REDUCTION PLANTS

Source: U. S. EPA 1980c

FIGURE D-9 LOCATION MAP OF THE PRIMARY ALUMINUM INDUSTRY IN THE UNITED STATES



Source: Brady (1974)

FIGURE D-10 EROSION MAP OF THE UNITED STATES: (1) severe sheet and gully corrosion; (2) moderate to severe erosion of mesas and mountains; (3) moderate to severe wind erosion with some gullying; (4) moderate sheet and gully erosion with some wind action; (5) moderate sheet and gully erosion locally; (6) erosion rather unimportant. [After U.S. Soil Conservation Service.]

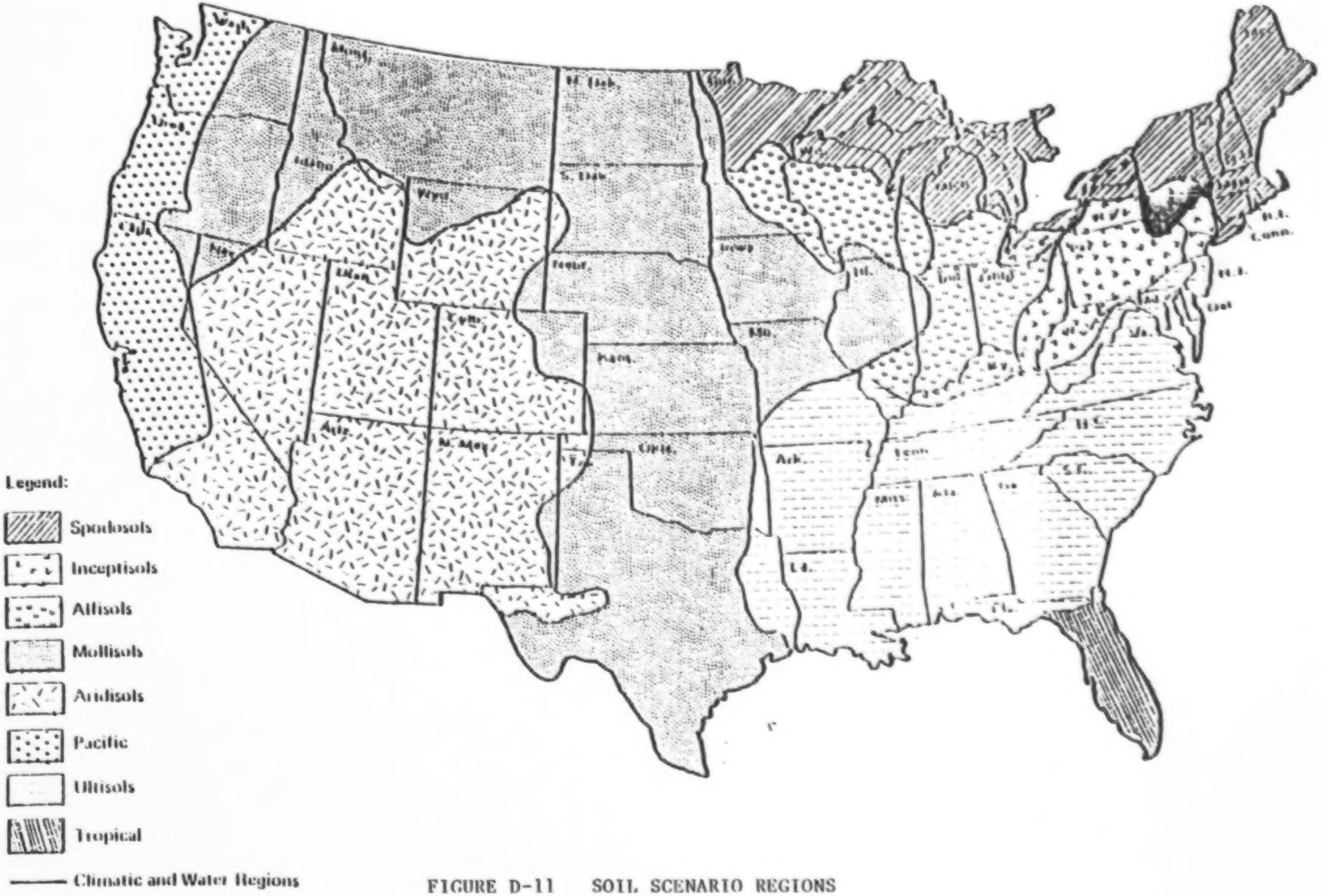

Legend:
Spodosols
Inceptisols
Alfisols
Mollisols
Aridisols
Pacific
Ultisols
Tropical
Climatic and Water Regions
Wash.
Oreg.
Calif.
Nev.
Idaho
Mont.
Wyo.
Utah
Colo.
Ariz.
N. Mex.
N. Dak.
S. Dak.
Nebr.
Kans.
Okla.
Tex.
Iowa
Mo.
Ark.
La.
Ill.
Ind.
Ohio
Ky.
Tenn.
Miss.
Ala.
Ga.
Fla.
S.C.
N.C.
Va.
R.I.
Conn.
N.J.
Del.

FIGURE D-11 SOIL SCENARIO REGIONS

Source: Brady 1974

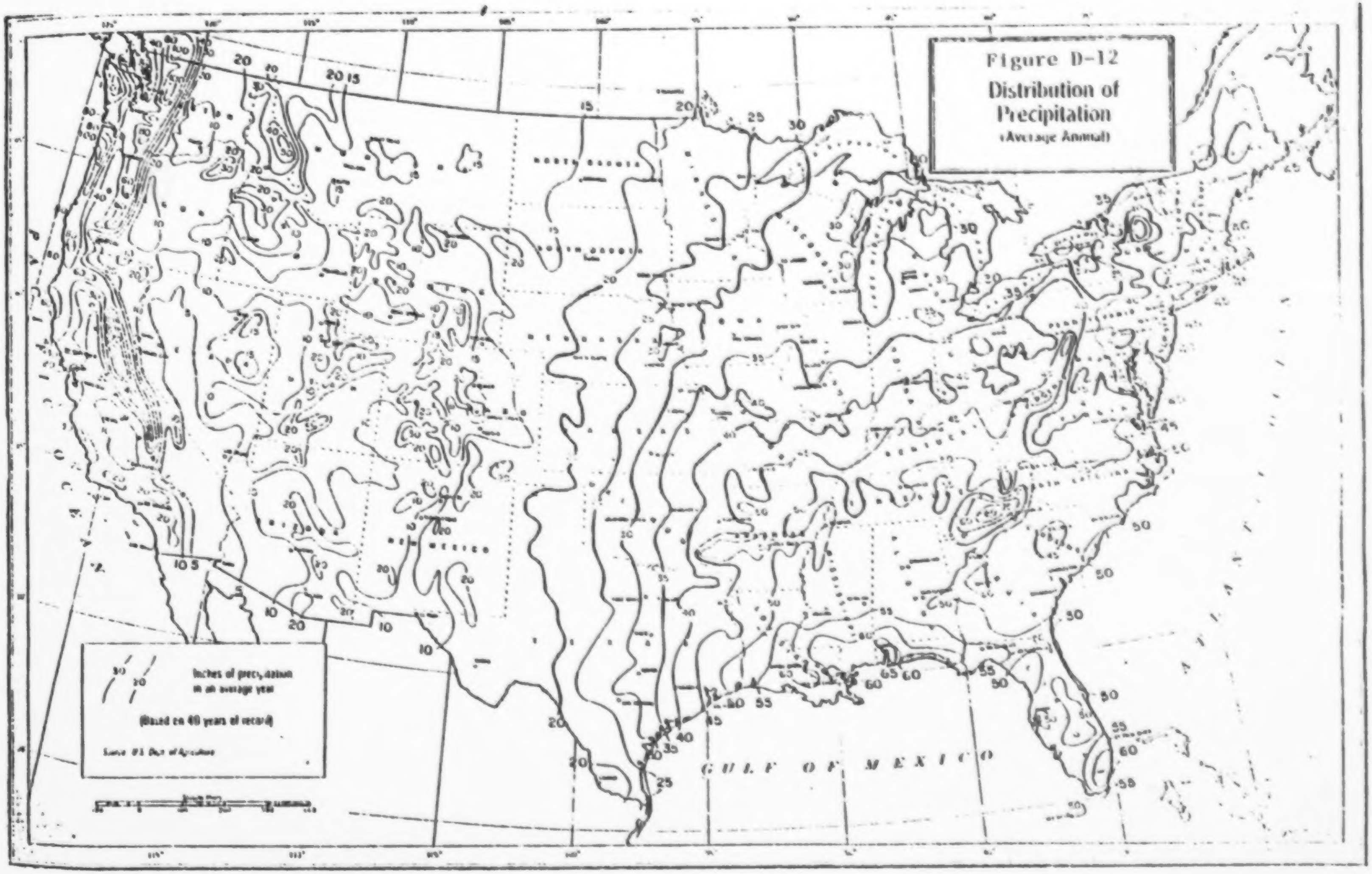


Figure D-12
Distribution of Precipitation
(Average Annual)

PACIFIC OCEAN
GULF OF MEXICO
Inches of precipitation in an average year
(Based on 40 years of record)
Source: U.S. Dept. of Agriculture

Source: Geraghty and Miller 1973

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TABLE D-1 ESTIMATED USES OF MSMA IN THE UNITED STATES BY REGIONS AND CATEGORIES, 1979 (kkg Arsenic Equivalent)

	Category				
Region	Agriculture	Industrial/ commercial	Government agencies	Home garden	Total
Northeast	Negl.	83	28		
North Central	139	194	55	Geographic distribution not known	
Southeast	500	279	70		
South Central	3,531	473	70		
Northwest	56	111	27		
Southwest	84	111	83		
Total	4,310	1,250	330	510	6,400

Source: U. S. EPA 1980c

B6
10

TABLE D-2 COMPANIES ENGAGED IN BORON PRODUCTION

Company and Location	Annual Capacity	Capabilities
Kerr-McGee Corp. Kerr-McGee Chemical Location, Subsidiary Searles Lake (Trona) CA	100	Borax decahydrate, borax pentahydrate, anhydrous borax, boric acid
Searles Lake (West End), CA	25-30	Borax decahydrate, borax pentahydrate, anhydrous borax
Stauffer Chemical Co. San Francisco, CA	Unknown	Unknown
Texas United Corp. American Borax Corp.,Subsid. Furnace Creek District, Inyo City, CA	50-75	Colemanite, alexite/probertite
U.S. Borax Chemical Corp. Boron, CA Wilmington, CA	700	Refined borax decahydrate, crude and refined borax pentahydrate, oxide and refined anhydrous borax

Source: U. S. EPA 1980c